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SYMPOSIUM

LUNG ABSCESS. SOME ASPECTS OF ETIOLOGY AND MEDICAL TREATMENT.*

DR. JAMES ALEXANDER MILLER, New York.

ETIOLOGY.

The generally accepted idea of the etiology of pulmonary suppuration has been that it follows certain cases of pneumonia in which, because of certain conditions in the lung little understood, necrosis and suppuration result instead of the more usual process of resolution.

Certain etiological factors have been recognized as especially favoring the development of pulmonary suppuration, such as the inhalation of foreign bodies, operations upon the respiratory tract, especially tonsillectomy and the extraction of teeth, and less frequently following operations upon suppurative foci in more distant parts of the body. In all of these conditions the conception has been that pneumonia first occurred and that then later following the pneumonia there was suppuration.

Dr. Adrian Lambert and I, in studies of a considerable series of abscess of the lung, were impressed by the fact that a very considerable percentage of the cases apparently occurred without any evidence of either pneumonia or of any of the predisposing factors above mentioned. The fact, also, that in cultures made from these

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lung abscesses very frequently none of the ordinary pathogenic bacteria could be found, although the abscesses swarmed with anaerobic bacteria, led us to suspect that possibly the anaerobic organisms played a real pathogenic role in the formation of the abscess instead of simply the secondary one which had been ascribed to them.

It is significant also that the anaerobes found in abscesses correspond to a group frequently found in persons with bad oral hygiene, particularly diseased tonsils and pyorrhea alveolaris.

Since that time a considerable amount of experimental evidence concerning the role of anaerobes in the formation of abscesses has been elicited. Klein and Smith have produced pulmonary abscesses in animals by intratracheal injections of the scrapings from infected teeth.

Crowe and Scarf injected the sinuses of dogs with such scrapings and obtained abscesses of the lung in two out of six cases.

Fetteroff and Fox found infected emboli in the bed of removed tonsils.

Cutler and Schueter were able to produce pulmonary abscesses by placing emboli infected with anaerobes in the jugular vein of animals.

From these experiments it would appear that abscess of the lung may be produced either by inhalation of infection or through the blood stream by embolus from more distant parts of the body. It also appears to be proved that the anaerobic bacteria frequently found in the mouth will produce abscesses of the lung experimentally under certain conditions.

The anaerobes which have been found most commonly, both in dirty mouths and in abscesses of the lungs, are some of the spirochaetes, fusiform bacilli, vibrios, motile bacilli of the colon group and anaerobic streptococci.

The important practical application of these newer ideas of the possible role of the anaerobe in the production of pulmonary abscess is the fact that operative procedures, especially tonsillectomy and tooth extraction in the mouths of persons with poor oral hygiene, produce ideal conditions for the transmission of these organisms to the lungs and points to the extreme importance of cleaning up the mouth very carefully prior to any such operation. It would appear that as a preventive procedure this is most important and is probably more so than the much discussed problem of the relative danger of local and general anesthesia for such operations.

MEDICAL TREATMENT.

Early abscess of the lung should be considered primarily as a medical disease. Chronic pulmonary abscess usually calls for surgery.

Rest and Posture: It is our experience that rest, combined with proper postural treatment, is the best method to be employed first in the treatment of early pulmonary abscess. From 45 to 50 per cent of early abscesses recover by this means alone. It has long been known that a certain number of abscesses of the lung cure themselves completely by spontaneous rupture and evacuation. It is the object of this medical treatment by rest and posture to increase the number of such cures and the advantage to the patient of avoiding operation, with a very long convalescence and frequent surgical dressings, to say nothing of the danger of the operation itself, is very evident.

We have also found that surgical operation during the acute phases of abscess of the lung carries with it a very high mortality, 65 to 70 per cent. By careful, frequent X-ray examinations we believe it is possible to gauge the point when this acute inflammation about the abscess is subsiding and when such X-ray studies are associated with frequent surgical consultations, as they should be, it is usually possible to judge of the phase of the disease in which operation can be performed with the least risk and with the best hope of success.

On the other hand, if the lung lesion does not clear up satisfactorily after one or two months of this conservative treatment, we have found that it probably will not and that then surgery should be called upon. If this medical treatment is persisted in too long, the case drifts into the chronic stage and operation then again becomes more dangerous and complete cure is less apt to occur, because of the liability to permanent bronchial fistula or to the failure of the abscess to close completely on account of the rigid fibrous walls which have been allowed to form.

Medical care, therefore, consists mainly in insisting upon rest and postural drainage during the early acute stages, and later consists in the exercise of proper judgment as to whether this treatment alone is likely to succeed, rather than to allow the case to become too chronic and less favorable for the surgical operation which will eventually be necessary.

Bronchoscopy: In association with rest and posture, we have used bronchoscopy as an aid in the treatment of certain cases in order to assist drainage. We usually employ this method in cases which do not respond in a few weeks to rest and posture alone before resorting to more radical surgery. When foreign body inhalation is suspected or possible, bronchoscopy is indispensable, both for the diagnosis and for the cure by removal of the foreign body.

On the other hand, we believe that the practice of long-continued bronchoscopy over many months is mainly palliative and has the

same unfavorable effect as does the long-continued medical treatment, allowing the case to become so chronic as to be surgically unfavorable.

Artificial Pneumothorax: Artificial pneumothorax has been advocated by some as an ideal method of treatment for abscesses of the lung. We have used this method in a considerable number of cases and found it very unsatisfactory, and sometimes dangerous. If the abscess of the lung is situated near the periphery the introduction of air into the pleural cavity may result in the tearing of adhesions, with the rupture of the abscess into the pleural cavity and the occurrence of a very fulminating empyema, which is generally fatal.

On the other hand, the air introduced by the method of artificial pneumothorax cannot be exactly controlled and sometimes it will get in between the lung and the mediastinal pleura, resulting in a cutting off of the drainage rather than in favoring it. We believe that the principle of artificial pneumothorax as it is used in pulmonary tuberculosis is mainly for immobilization of the infected organ. The principle of treatment in pulmonary abscess, on the other hand, is drainage, which is by no means always favored by this method.

SUMMARY.

1. Abscess of the lung is frequently caused by the post-operative infection from infected mouths.

2. Cleanliness of the mouth is a very important prophylactic measure in avoiding post-operative pulmonary abscess.

3. The diagnosis of pulmonary abscess is comparatively simple and depends largely upon X-ray evidence.

4. The treatment of acute pulmonary abscess is primarily medical by rest and posture.

5. Bronchoscopy is a valuable aid in some cases and is indispensable in cases due to the inhalation of foreign bodies.

6. Medical treatment and bronchoscopy should not be continued so long that the case becomes chronic.

7. Surgery is necessary in about 50 per cent of the cases and if employed at the favorable stage is not attended with serious risk. It is usually the only treatment for chronic cases.

8. The close co-operation of nose and throat surgeon, the internist, the surgeon and the bronchoscopist is essential to obtain the best results.

LUNG ABSCESS. ROENTGENOLOGIC DIAGNOSIS.*

DR. LEON LE WALD, New York.

This presentation was in the form of a series of Roentgen pictures to illustrate the points in question. Unfortunately, it is impossible to reproduce Dr. Le Wald's contribution to the Symposium.

BRONCHOSCOPIC DIAGNOSIS AND TREATMENT OF LUNG ABSCESS.*

DR. SIDNEY YANKAUER, New York.

I have been asked to speak on the subject of "The Bronchoscopic Diagnosis of Lung Abscess". This involves not only the study of the disease itself, but also, what is more important, its differential diagnoses from other diseases of the lungs, and therefore a recognition of these diseases and of the difference between them. In a presidential address which I delivered last spring, before the American Bronchoscopic Society, I urged upon my fellow bronchoscopists the need of more energetic development of this phase of bronchoscopic work. Before a body of men who are interested in the broader field of general medicine, and who may or may not have followed the progress of bronchoscopy, I may be permitted to state and to emphasize the fact that there is such a thing as bronchoscopic diagnosis of chest diseases, that there is a bronchoscopic symptomatology which is pregnant with interest and fruitful of results.

This bronchoscopic symptomatology is of especial interest to the general diagnostician for the following reasons:

1. Because in some cases it affords the only means of making a final and conclusive diagnosis, as for instance, because it affords the opportunity for a biopsy.

2. Because negative bronchoscopic findings, like other negative findings, make possible a diagnosis by exclusion, which is often valuable.

3. Bronchoscopy sometimes succeeds where other means fail, as by making possible a bacteriological study of the uncontaminated flora of the deeper parts of the lungs, even to the extent of finding tubercle bacilli when repeated examinations of the expectorated sputum have failed to disclose them.

4. Because rare and unsuspected conditions not otherwise recognizable, may be found.

5. Because in appropriate cases it becomes possible to institute therapeutic measures at the same time with, and because of, the recognition of the true condition which is actually present.

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After years of experience, including many striking cases of my own, as well as many brilliant successes of my colleagues, I can unhesitatingly declare that no diagnosis of an intrathoracic disease is scientifically complete which does not include a report of endoscopic findings and that the neglect of an endoscopic examination is a failure to avail ones self of the full possibilities of medical science.

It is indeed true that many chest cases are too sick to be bronchoscoped, but the fear of bronchoscopy which still lingers in the minds of many is no longer justifiable. Bronchoscopy in its early days had its mishaps, and even its fatalities. Like other diagnostic measures of surgical or semisurgical nature, these were due to the ignorance and to the lack of skill which was unavoidably associated with their newness. Bronchoscopy, however, is no longer done at random by anybody. The novice is required to learn at the feet of mature and experienced teachers; the roadway has been cleared for him and he is forewarned of the dangers which he must meet. In the hands of the experienced and skillful bronchoscopist, who is familiar with its possibilities and its limitations, bronchoscopy today is practically devoid of danger.

On the other hand, it must not be supposed that a skillful technician is necessarily a skillful diagnostician. Diagnostic acumen comes from experience, and experience comes from repeated observation and study of actual cases. The patients are in the hands of the internist and under his complete control. Every modern, completely manned hospital has, or should have, a bronchoscopist on its staff. Unless the bronchoscopist is given the opportunity to examine and study the bronchoscopic appearances in all kinds of chest diseases, he cannot be expected to be familiar with them. If, therefore, the internists desires to avail themselves of this truly wonderful means of diagnosis, it behooves them to develop their own bronchoscopic diagnostician, by affording their own bronchoscopist the opportunity presented by the host of clinical material which lies in their own hands. Sincere co-operation between the two groups of men is essential. The confidence of the internist in the value of bronchoscopic diagnosis will grow with the increasing experience of the bronchoscopist and the lives that will be saved and the lifetimes of suffering that will be prevented will be ample justification and reward.

During the past winter season there were referred to my department for bronchoscopic diagnosis 100-odd patients from the general services of Mt. Sinai Hospital. These cases may be described colloquially as so-called obscure chest cases and a bronchoscopic diag-

nosis was considered necessary, either because the data obtained by other means were insufficient to make a satisfactory diagnosis, or because it was desired to verify and to afford conclusive proof of diagnoses already suggested by other methods of examination. These cases do not therefore include any cases in which tubercle bacilli were found in the expectorated sputum and only a few with positive Wassermann reactions.

The first group of cases of special interest and importance were five cases in which foreign bodies were found in the bronchi. These were all cases in which the history of inhalation of a foreign body was absent, and in which the radiograph did not show the presence of a foreign body in the bronchi. This group therefore does not include two cases in which there was no history of inhalation, but in which the radiograph did show the presence of a foreign body. In other words, these foreign bodies were all radio-transparent. This proportion of unsuspected foreign bodies is rather large. One of the patients, a child, died two weeks after the removal of the foreign body from cerebral hemorrhage, but the rest recovered. One of them was of special interest; this was a girl, age 25 years, who was sick for five years and then developed an empyema, for which she was operated upon. As the fistula did not heal, a more extensive operation was contemplated and a preliminary bronchoscopy was ordered as a routine procedure. The wisdom of this practice was amply demonstrated by the complete recovery of the patient after the removal of the foreign body.

Unsuspected foreign body must not be overlooked, even when the history of post-tonsillectomic lung abscess is clear, as is illustrated by the following cases (which, however, do not belong in the same series of cases).

Case 1: A patient with such a clear history of post-tonsillectomic abscess that preliminary bronchoscopy was omitted, was operated upon by the surgeon. The patient died after the first stage of the operation and autopsy disclosed the presence of a nut shell in the bronchus. The true history of the case was probably as follows: A cough, caused by the foreign body, whose inhalation was forgotten or ignored, was erroneously attributed to the tonsils. The irritation of the general anesthetic precipitated the symptoms of lung abscess.

Case 2: A woman with a clear post-tonsillectomic history was bronchoscoped as a matter of routine. A piece of bone was found in her bronchus. Subsequent inquiry elicited the fact that at the time of the tonsillectomy, a piece of bone was removed from her nose.

The piece of bone removed from the bronchus had the size and contour of the anterior end of the lower turbinate bone.

Case 3: A boy developed a spasmodic cough, with expectoration, after tonsillectomy. A piece of rubber tubing, such as is inserted into the nostril by anesthetists for intrapharyngeal anesthesia, was cut off by the snare and found its way into the bronchus.

Among the important services which the bronchoscope can render to the internist is the diagnosis of malignant diseases of the lung. While it is often possible for the internist to make this diagnosis by inference, a positive conclusion cannot be reached until a biopsy has been performed. The majority of cases of carcinoma of the lung involve the lumen of the bronchial tree, either because they begin in the mucous membrane of the bronchus or because they eventually break through the bronchial wall and become visible within the bronchial lumen. The classical picture, therefore, is one in which the history, symptoms, physical signs and radiographic picture point to the possibility of malignancy. The bronchoscope brings the growth directly into view and makes possible the removal of a specimen. There were 14 such cases in this group, and this experience is so familiar to bronchoscopists that we feel that the possibility of making a diagnosis in this manner is no longer news to any well informed physician.

There were, however, three cases in which all signs pointed to a malignant growth, but bronchoscopic examination was negative. In one of these, which came to autopsy, a carcinoma was found which involved only the periphery of the lung and did not extend into the bronchial lumen.

The age of the patients in relation to the diagnosis of malignancy is no different in the lung than elsewhere. There were only three cases less than 40 years of age, one of 34, one of 33, and one of 26. In the early stages of lung carcinoma, the symptoms are hardly distinguishable from those of the simple lung abscess. But though lung abscess usually begins in early adult life, many of the patients live until they are long past the critical age. What is therefore of interest is not the age at which the patient is first seen, but the age at which his symptoms began, and it is significant that of all the patients in this series, who began to have symptoms of pulmonary suppuration after the age of 40, more than half were found to be suffering from a malignant growth. The other conditions which may cause symptoms in elderly people similar to those of lung abscess which have been met with in this group are syphilis, diabetes, actinomycosis and

unresolved pneumonia. When these conditions are excluded, as they generally can be, the proportion of malignancy becomes still greater.

There were two cases of sarcoma of the lung in the group. Sarcoma originates as an interstitial growth and may cause a distortion of the bronchial lumen recognizable endoscopically long before the growth has penetrated the wall of the bronchus and becomes visible in the lumen. All the cases of sarcoma of the lung which I have seen not only in this group but in previous experience, have been located in the lower lobe and this fact seems to be of some significance in diagnosis.

Of special interest to me have been a number of cases in which the bronchoscopy recognized a distortion of the bronchial lumen, apparently due to the presence of the mass outside of the bronchial wall. The pressure may vary from a moderate displacement of the bronchus to a distortion so pronounced as to almost completely close the entire lumen. I would prefer to reserve the term of stenosis of the bronchus to those cases in which the disease of the mucous membrane itself causes a narrowing of the lumen, such as inflammatory swelling, cicatricial contraction or tumor formation, because such cases present a picture in the bronchoscope which is entirely different from the narrowing, due to the pressure of a mass outside of the bronchus itself. Such pressure may be due to new growths, to enlarged glands, to cicatricial contractions and even to a neighboring lung abscess. Naturally, the extra-bronchial mass itself cannot be seen and the removal of a biopsy by cutting through an otherwise healthy bronchial wall would not be justifiable, but the location of the pressure is of diagnostic significance. For instance, as just mentioned, sarcoma causes a distortion of the lumen in the lower bronchi; enlarged glands in the hylum cause pressure in the neighborhood of the bifurcation of the trachea and, in fact, a change in the shape or contour of the spur at the bifurcation usually indicates the presence of an enlarged lymph node in the crotch of the bifurcation.

A dilated heart makes pressure upon the left bronchus from below. Aneurysm of the aorta may cause an indentation in the wall of the left bronchus, either in front, on the outer wall, or posteriorly, according to the location of the aneurysm. Sarcoma of the mediastinum usually arises in the posterior mediastinum and makes pressure upon the posterior wall of the trachea. Dermoid cysts occur in the anterior mediastinum and make pressure upon the anterior wall. Cicatricial contraction from a scar formation has been observed in connection with healing tuberculosis and the endoscopic picture is quite distinguishable from the picture caused by the pressure of a growth.

Benign tumors of the bronchial tree are occasionally met with. In this series a hemangioma and a papilloma were found, but I have seen and removed two fibromata from the bronchi in cases not included in this series. In both these cases the tumor produced a marked stenosis of the bronchus, with bronchiectasis and suppuration in the distal portion of the lung. One case of congenital stenosis of the right main bronchus occurred in this series. The stenosis did not become evident until dilatation and infection of the bronchi below the stenosis occurred. There was one case of pneumoconiosis which became evident endoscopically by the presence of an enlarged gland at the bifurcation of the trachea. There were two cases of simple ulceration of the trachea, causing cough and hemoptysis, which were promptly healed by topical application. There was one case of specific ulceration of the trachea and one case in which the tracheal mucous membrane resembled in appearance that of the laryngeal mucous membrane in lupus, and which was diagnosed as lupus of the trachea, even though the diagnosis could not be verified microscopically. There were two cases in which simple granulomata were present, which upon removal proved to be of tuberculous origin, even though the diagnosis of tuberculosis was not made beforehand, repeated examinations for tubercle bacilli having been negative. There were three cases of unresolved pneumonia which are of interest because all three yielded promptly to a short series of endoscopic treatments consisting of aspiration and lavage of the bronchi. The mechanical condition which has been found in these unresolved pneumonia cases was the presence of plugs of inspissated secretion in the lumen of the smaller bronchi and it was the removal of these plugs which resulted in the prompt cure of these cases. The internist is apt to be extremely conservative in handling these cases because he knows that nature eventually will heal some of them, but it is nevertheless true that the early removal of the obstructing plugs will be of the greatest assistance to nature in bringing about this desired end. I am satisfied that when evidence of resolution does not follow promptly after the crisis of pneumonia, the sooner the patient is submitted to endoscopic treatment, the quicker recovery will take place.

As stated above, cases of frank tuberculosis were excluded from bronchoscopic examination; nevertheless, in 52 of this series of cases, hemoptysis was a prominent symptom. Hemoptysis occurred in about 75 per cent of lung abscess, or carcinomata, and of foreign bodies. The fact that hemorrhage is such a constant symptom in foreign bodies is of diagnostic interest, and deserves to be borne in mind. Experience has shown that hemoptysis, unless frequent and

profuse, is not a contra-indication to bronchoscopy, but that, on the contrary, in the absence of tubercle bacilli in the sputum, hemoptysis is a symptom that demands bronchoscopic investigation to determine its cause. This attitude is supported by the fact that there were two cases in which simple erosions were found to be the cause of hemoptysis and that the blood ceased to appear after the erosions were healed. In addition, hemorrhages occurred in such cases as papilloma, sarcoma, lupus, etc.

Among the cases studied were 45 cases which were diagnosed as bronchiectasis or as lung abscess. Whether, or how often these terms describe separate diseases existing separately in different patients is not clear to me.

Pathologically, a case of bronchiectasis is a case in which either one or more of the large bronchi is dilated, or in which there are several smaller branches showing dilatation, while an abscess presupposes destruction of tissue, whether through simple ulceration or through destruction of larger areas.

Bronchoscopically, a case of bronchiectasis is a case in which the visible bronchi are dilated or elongated. These may belong to the first, second, third, and sometimes the fourth order. An abscess is recognized bronchoscopically when pus continues to discharge from one or more branch bronchi, after repeated removal by aspiration. In this way only can the bronchoscopist determine that there is a reservoir of pus beyond the discharging bronchus. He cannot, unaided by other diagnostic measures, such as lipiodol injections, determine whether all the discharging bronchi lead to one cavity, or whether each leads to a separate cavity, or whether the cavities are epithelialized or granulating.

The roentgenologist recognizes a bronchiectasis when he can see the dilated walls of the bronchi, especially with the aid of lipiodol, but when he sees a cavity with a fluid level, it becomes an abscess.

When the surgeon opens a cavity in the lung, if he finds its walls smooth, and epithelialized, it is a bronchiectasis, whether the cavity is simple or multilocular. But if the cavity is lined with granulation tissue, it becomes an abscess, and sometimes he compromises with himself by calling it a bronchiectatic abscess.

Each observer emphasizes a distinction which is valid from his standpoint, but in actual practice there are few cases in which ulcerated cavities, confluence of neighboring bronchi, and dilatation and elongation of other bronchi are not associated in the same case.

Perhaps some of my hearers will be able to throw more light on this distinction than I can.

Mention has already been made of the use of lipiodol for mapping out the structure of the lung. When this procedure was introduced, such large quantities of the oil were deemed necessary as to give a picture of the bronchial tree consisting of solid black lines, representing the entire thickness of the bronchi. In the region of the smaller divisions, however, these shadows overlapped to such an extent as to obscure and confuse the picture, and to lead to erroneous conclusions. Entirely aside from the occasional iodine poisoning accompanying the use of large quantities, smaller quantities have the advantage of delineating the bronchi as fine parallel lines. In this way their size, shape and direction are fully shown, but in addition, overlapping bronchi can be recognized and distinguished from bronchiectasis, details of the lumen can be appreciated, and, what is most useful, stereoscopic views can be obtained. The exact amount which should be used in any case cannot be stated beforehand. It is therefore necessary to make the injection under the guidance of the fluoroscope, so that the injection can be ended when the necessary amount has been introduced. This is readily accomplished if the lipiodol is injected through a tube introduced through the cocainized larynx. Both rigid and flexible tubes have been devised for this purpose. The lipiodol can be made to enter any desired lobe by placing the patient in the optimum position for that lobe. It is also desirable to avoid moving the patient after the injection, or even changing his position until the picture has been taken.

When finer details are needed, as for instance, in determining the relation of a single branch bronchus to a cavity, tumor or foreign body, the lipiodol must be injected through a cannula introduced through the bronchoscope.

Different observers vary so widely in their technique as to preclude the possibility of comparing results, and greater uniformity is desirable. I would therefore propose the following postulates as the first step towards standardizing the procedure:

1. The injection of large quantities, showing the bronchi as broad black bands, must be regarded as a technical error. Only enough should be injected to outline the walls of the bronchi, without filling the lumen.

2. The injections should always be made under the guidance of the fluoroscope.

3. Injections through the bronchoscope are needed only in exceptional instances.

The bronchoscopic treatment of lung abscess is carried out under two different plans.

When the cavity can be entered with instruments through the bronchoscope, an attempt is made to enlarge the passage by dilatation. A small bronchus, normally about 3 m.m. in diameter, can be forcibly dilated to a diameter of about 9 m.m. in several sittings. Whenever such a large and free passage can be provided, a cavity in the lung cannot continue to exist—its contents are discharged and it collapses. The dilatation of the entering bronchus is followed immediately by marked and decisive improvement in the patient's condition.

It has been assumed by some that an abscess located near the hilus is the type which is amenable to such treatment and that an abscess situated near the chest wall must be operated upon surgically. Experience has shown, however, that it is not the location in the chest cavity which is the determining factor, but rather the point of entry of the entering bronchus. If the bronchus from which pus is coming is found to enter the abscess cavity on the proximal side, so that bronchoscopic instruments can be made to enter the cavity, a cure may be hoped for, even if the bulk of the abscess cavity be located near the chest wall. On the other hand, it may be impossible to carry out the intended manipulations. When this procedure is found impossible the bronchoscopist must adopt the slower plan of controlling the abscess by checking the advance of the pneumonitis which surrounds the abscess cavity itself. This is accomplished by endobronchial lavage and aspiration. This method is slow and laborious; many and frequent bronchoscopies are needed, extending over a period of many months.

In nearly all cases important and valuable results are obtained. The objectionable odor of the sputum disappears, its quantity diminishes, the periods of exacerbation are lessened, and the general well being of the patient is improved to such an extent that he becomes able to resume his occupation and to earn his livelihood.

In a few of the cases permanent cure was accomplished even by this method of treatment. In fact, the first case of lung abscess which was ever cured by bronchoscopic lavage was treated in this manner. This was a case of post-tonsillectomic lung abscess in a girl, age 13 years, who came under my care one year after the tonsil operation, in September, 1916. She was under treatment for nine months, having been bronchoscoped during that time 33 times. At the end of this period, June, 1917, she was entirely cured, clinically, bronchoscopically and radiographically, and she has remained well since. She has taken a general anesthetic for an appendectomy without any deleterious effect.

121 East 60th Street.

SURGICAL DIAGNOSIS AND TREATMENT OF LUNG ABSCESS.*

DR. ADRIAN V. S. LAMBERT, New York.

It is a pleasure to appear here and have the opportunity of saying the last word in the treatment of lung abscess at the end of a long program. The title of these remarks should be the role of the surgeon in the treatment of lung abscess, rather than the surgical treatment, because I imagine few in this audience are interested in the minute details of surgical technique.

The mere fact that I am asked to speak at all is an encouraging sign and would seem to indicate that, after all has been said and done, there is still a place for the surgeon in the cure of this condition. To many the role of the surgeon in the treatment of lung abscess means simply the drainage of a suppurative focus in the lung through an opening in the chest wall. To my mind, however, it is far different from that.

Lung abscess differs from other suppurative conditions, in that there is a proper time in its course when a case should be operated on, and there is an improper time. To undertake this too early is extremely hazardous, and to delay it too long greatly jeopardizes the patient's chance of recovery. The decision as to the optimum time for operation should depend on an accurate conception of the pathological processes taking place in the lung, and the ability of interpreting the X-ray plates in terms of those processes.

Operation undertaken during the early stages, while an acute, widespread exudative inflammation about the central necrotic focus is present in the lung will give a mortality of about 70 per cent. The reason for this is because drainage by incision, with a resultant bronchial fistula of the central necrotic focus, prevents drainage of the large portion of the lung involved in the widespread exudative process. With the establishment of a bronchial fistula the air rushes through it during the act of coughing and the lung remains almost stationary, the air vesicles filled with highly infectious exudate are not emptied of their contents and a widespread extension of the

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pneumonitis results, which accounts for 70 per cent of the cases ending fatally.

Under postural drainage and bronchoscopic treatment, this area of exudative pneumonitis gradually diminishes as the virulence of the reaction subsides and there remains the central necrotic focus, surrounded by a group of collapsed air vesicles, which drain directly into the abscess cavity. At this stage the X-ray shows a more or less discrete, dense shadow representing this zone of collapsed vesicles surrounding an area of lesser density corresponding to the central necrotic focus. We have found that these collapsed vesicles will retain their respiratory epithelial lining cells about two or three months and that during this period the vesicles are capable of being reaerated and of returning to a normal condition. This period then is the ideal time in which to drain an abscess by an open incision through the chest wall. The process is well localized, the incision will drain completely the entire area infected and the passage of air through the fistula, by its oxygenating power, aids the patient in combating the anaerobic infection which is always present. The danger of establishing a permanent bronchial fistula is small; the necessity of having to do so is almost *nil,* and although the period of convalescence is a long one there is a 90 per cent chance that the patient will be restored to perfect health.

If, on the other hand, a patient is watched and treated by conservative treatment without any improvement, but remaining stationary, whether it be by postural drainage, by bronchoscopy or other methods, profound changes take place in the air vesicles surrounding the central area, due to the presence of prolonged infection and suppuration. There is a pronounced increase in the connective tissue elements in their walls, they gradually but inevitably lose their respiratory epithelial lining cells and there results a coalescence of their walls, with the resultant obliteration of the air spaces, and there is formed a dense connective tissue zone about the central cavity, which is rich in blood vessels of large calibre, having thin, friable walls. This differs widely from the thick nonvascular reaction which takes place in the pleura under the presence of prolonged suppuration. To await until this occurs before operating increases the immediate operative risk enormously. The most frequent causes of death in these cases are hemorrhage and cerebral embolism, both of which are directly due to the dense noncontractile character of the connective tissue wall which tends to hold open the mouths of the injured blood vessels. In these cases also there often arises the necessity of establishing a permanent bronchial fistula, because the

anaerobic bacteria can and often do continue to live in this dense connective tissue for many months, if not years, and if one permits the fistula to close, a reappearance of the abscess may be anticipated

If in the course of his disease a patient, after a period of improvement, both in symptoms and X-ray findings, suffers a relapse or has a severe hemorrhage, I am convinced that he should have an open drainage, after he has recovered from the acute stage of this relapse. It is, indeed, true that certain of these cases may ultimately recover on conservative treatment, but their number is small and one runs a greater risk in allowing them to drag on week after week, with the possibility of another exacerbation than is involved by an open drainage.

There are several other points of a surgical nature which should be of interest to this audience. The exact localization of an abscess is one of the most difficult problems with which a surgeon has to deal, and often too little attention is given to this phase of the problem. It is not enough simply to designate in which lobe the abscess is situated, important as this certainly is, but one should visualize its situation in terms of a cross-section of the thorax and determine, if possible, the exact position on the chest walls where the abscess is nearest to the parietal pleura. In our experience the use of the fluoroscope is the most helpful aid we have, where by rotating the patient we gain more information than by any number of plates.

I know of no way by which we can determine the presence of adhesions between the visceral and parietal pleura except by the use of pneumothorax, and this carries with it too great a danger for us to employ it for this purpose. It has been our experience, however, that the longer an abscess exists, the more circumscribed and more dense the adhesions become. Since it is not possible to surely diagnose the presence of adhesions we are convinced that it is never justifiable to needle an abscess through the chest wall. We have seen some dire results follow this procedure, where in the absence of adhesions a fatal empyema has supervened. The only safe procedure is to cut down on and remove a portion of a rib, examine the pleura and demonstrate the presence of adhesions and then locate the abscess by a needle. If adhesions are absent, the wound must be packed and the formation of adhesions awaited before undertaking further manipulation.

To recapitulate, then, the role of the surgeon implies not only the operative technique of open drainage, but also the courage to refuse to operate too early, to insist on when the proper time has arrived for operation and to warn all concerned that a prolonged delay, with a

temporizing policy, carries with it grave risks. He should be consulted early and be afforded the opportunity of following the progress of a case and there should exist a close co-operation between everyone: the internist, the bronchoscopist, the radiologist and the surgeon. The day has passed, at least in this condition, when the surgeon can disregard the use of the stethoscope and the significance of physical signs in the chest, and the time should have passed when the surgeon is called in as a kind of exalted carpenter, a history related to him, a diagnosis given to him, and he be then walked up to a patient, shown his chest, with an ink mark made over some rib or other, and told, "There's the abscess. Open it", and then have the surgeon meekly and calmly go ahead.

Every case requires mature judgment and the opinion of the surgeon familiar with the physical conditions as found at operation should be sought and he should not be looked upon as a necessary evil and last resort in a desperate situation.

CONTRIBUTION TO THE SURGICAL INDICATIONS IN PURULENT LABYRINTHITIS AND OTITIC MENINGITIS.*†‡

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PART I—LABYRINTHITIS.

Before entering into a discussion of the treatment of acute labyrinthitis, it is essential to differentiate sharply the various types and describe their etiopathogenesis. We feel that to accept the terminology of serous and purulent, circumscribed and diffuse labyrinthitis, is insufficient in itself as a guide to determining therapy.

To begin with, let us recall that a localized purulent focus will cause an inflammatory reaction in the adjacent parts. It is therefore possible that a localized purulency in the labyrinth will produce an inflammatory reaction in the remainder of the labyrinth. The diffuse labyrinthitis which results is of a serous nature and may be termed a diffuse serous labyrinthitis. Nevertheless, the pathological lesion which produced this serous labyrinthitis is a purulent focus within the labyrinth. In other words, there is associated with a circumscribed purulent labyrinthitis a serous labyrinthitis of a diffuse nature. If this is clear, it is also clear that one must then classify as purulent labyrinthitis not only the diffuse purulent labyrinthitis following meningitis, trauma, spread of infection from the tympanomastoid and gross necrosis due to embolus or thrombus of the labyrinthine vessels, but also the circumscribed lesions of the outer capsule which result from purulency in the tympanum and mastoid process. As concerns serous labyrinthitis, if we disregard the type which occurs in mumps, scarlet fever and other toxemias, we are confronted solely with that type which occurs as a reaction to a neighboring suppurative process. A serous labyrinthitis may ensue in the course of an acute purulent otitis media, an acute mastoiditis or a chronic mastoiditis of the dangerous type. It may also follow in the course of a rapid erosion of the outer capsule, where the aqueduct channels have not had opportunity to accommodate themselves sufficiently to the excess of fluid in the perilymphatic space. In such

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an instance, it is possible for a serous labyrinthitis to cause complete destruction of the function of the inner ear by pressure necrosis alone.

Clinically it is impossible to differentiate between a diffuse serous and a diffuse purulent labyrinthitis. Both present an exceedingly stormy picture at the outset. Spontaneous nystagmus, vertigo, vomiting, labyrinthine ataxia and the forced position are present in both. Both may follow a chronic suppurative lesion of the middle ear and both show a gradual cessation of the symptoms as the lesions progress either toward recovery or toward destruction of function. Both exhibit a high temperature. During the early acute stage, the caloric tests will reveal a hyperstimulation of the end organ. While it is true that a purulent labyrinthitis will always destroy cochlear and vestibular function, it is equally true that a serous labyrinthitis of sufficient extent will also destroy function if the intralabyrinthine pressure becomes too great. Consequently our functional examination is not without its limitations in differentiating the diffuse suppurative from the diffuse serous labyrinthitis.

Our main concern in the surgical types of labyrinthitis is the possibility of the spread of a purulency into the subarachnoid space through the internal auditory meatus, the aqueductus cochlearis or the ductus endolymphaticus. It has been determined by clinical observation that the surest guide to the type of diffuse labyrinthitis is the reaction of the cerebrospinal fluid. In a purulent labyrinthitis the spinal fluid will show definite evidence of a threatened invasion of the subarachnoid space, if a purulent meningitis does not already exist, manifested as a meningitis sympathica. There are increased cells in the fluid—largely polynuclear, increased pressure, cloudiness, traces of albumin and sometimes globulin, a normal sugar content and no bacteria. Where such a fluid is found in the presence of a clinically diffuse labyrinthitis, one may be certain that there is suppuration within the labyrinth. On the other hand, where a normal spinal fluid with but a slight increase in pressure is found, the labyrinthitis may be considered of a serous nature. If, on successive examinations, such a normal fluid is found to change in nature and assume the characteristics before described, it is indicative of an extension of the circumscribed purulent labyrinthitis beyond the protective zone of granulations and an infection of the remainder of the labyrinth.

Where no threat to the meninges exists, it is our contention that no surgery on the labyrinth is required. Where a meningitis exists as the causative factor in the diffuse purulent labyrinthitis—in other

words, where the lesion in the inner ear is secondary to a purulent leptomeningitis — surgery on the labyrinth *per se* is not indicated. That such a condition is possible is demonstrated repeatedly during the course of epidemic meningococcus meningitis. Where a circumscribed purulent labyrinthitis exists, we feel that the removal of the causative factor is sufficient to stop the progress of the lesion in the outer capsule and that, with the removal of the irritant, nature will heal the labyrinthine lesion and prevent further extension. So, in the diffuse serous labyrinthitis, the removal of the causative factor in the mastoid and middle ear will result in a rapid cessation of the disease.

In the event that there is found in connection with a diffuse labyrinthitis a definite reaction in the meninges which points to a threatened intrameningeal invasion, it is our contention that a labyrinthectomy alone is not sufficient. It is also essential to drain the lesser cisterns, as recommended in the second section of this paper. We reason as follows: In the event of a purulent diffuse labyrinthitis with threatened meningeal involvement, surgery limited to the labyrinth alone may cause a more rapid spread of the infection into the subarachnoid space by virtue of the traumatic disturbance of the limiting protective forces. We feel, therefore, that it is far better to drain the meninges early than to await the appearance of a suppurative meningitis.

The following cases are presented in substantiation of our contentions. The first two were cases of circumscribed purulent labyrinthitis which cleared up without surgery on the labyrinth. The third case was one wherein complete destruction of the labyrinth was found at operation, but wherein no symptom was at any time presented to warrant suspicion of such an extensive lesion. The last case was one of diffuse serous labyrinthitis following a radical mastoid operation, wherein destruction of the labyrinthine function ensued, but no surgery on the labyrinth was attempted.

Case 1: Male, age 24 years, was first seen by us on May 10, 1926. He complained of a discharge from the left ear for 19 years. Examination of his ear showed a large marginal defect in the left drum and a foul discharge in the left middle ear. Functional examination of his labyrinth revealed normal reactions to the rotation and the caloric tests. The audiogram showed a loss of hearing in the left ear. He was advised to have a radical mastoidectomy performed but refused. He was seen again by us on June 24, 1926, at which time the condition of his ear was the same.

One Dec. 10, 1926, he suddenly complained of severe headache on the left side of the head, severe vertigo and falling to the left. Otoscopic examination showed a profuse, pulsating discharge from the left ear. The mastoid process was tender. Functional examination of the labyrinth revealed a positive fistula test and exaggerated responses from the caloric test (the nystagmus appeared within 15 seconds of douching the left ear).

The patient was sent to Beth Israel Hospital, where a lumbar tap revealed fluid under normal pressure. The examination of the fluid gave normal findings. The blood count showed 4,200,000 red cells, 82 per cent hemoglobin, 12,000 white cells and 79 per cent polynuclears. The urine was normal.

A radical mastoid operation was performed on Dec. 11, 1926. The mastoid process was found eburnated. In the region of the antrum and filling the entire middle ear was a cholesteatoma. When this was removed by soaking it out with alcohol, an erosion of the horizontal semicircular canal was noted. On attempting to strip the matrix from the middle ear, the left side of the face was observed to twitch several times. Because of this, the operation was completed without entirely removing the matrix from the middle ear. The wound was left open and packed lightly with gauze soaked in alcohol.

The patient made an uneventful recovery following this procedure. All symptoms of headache and vertigo disappeared immediately after operation. A spinal fluid examination two days after operation was normal. On Dec. 21 the plastic operation on the canal was performed and flaps were cut after Panse's technique. The wound was closed completely. The after-treatment was uneventful. The patient has completely recovered and the radical cavity is dry.

Comment: In this case the patient was suffering from a localized purulent labyrinthitis due to erosion of the outer capsule. At operation the erosion was found to involve the lateral canal. This was not touched. Only the causative factor was removed and the lesion in the labyrinth allowed to heal.

Case 2: Male, age 36 years, was examined by us on July 25, 1926. He complained of an intermittent discharge from the right ear for seven years. For the two months prior to our seeing him, he complained of a slight pain over the right mastoid area. On July 13 he had a severe dizzy spell, with vertigo, vomiting and falling to the left. He had no headache. He stated that he did not hear with the right ear.

Otoscopic examination showed a large defect in the right drum. The middle ear contained cholesteatomatous masses. Functional ex-

amination of the hearing showed a marked loss in air conduction in the right ear for all forks. The Weber test showed lateralization to the right ear. Fistula test was positive. There were no spontaneous vestibular phenomena. The rotation test showed equal reactions after turning in both directions. The caloric test was not done. Radiographic examination of the mastoid process by Dr. Taylor revealed a sclerotic mastoid process with an area of destruction within it suggestive of a cholesteatomatous cavity. He was sent to Beth Israel Hospital, where a blood count, spinal fluid examination and urinalysis revealed normal findings.

A radical mastoid operation was performed on July 28, 1925. A large cholesteatoma was found which had entirely destroyed the posterior canal wall. The cholesteatomatous masses were dissolved out with alcohol and the matrix was then stripped off. The facial nerve was found exposed by the disease in the horizontal and descending portions. The horizontal canal was covered with granulations, which indicated the site of the fistula. The wound was left open. The patient reacted favorably for the next week and on the eighth day after operation the wound was closed and the Panse operation on the canal was performed. An uneventful recovery ensued. The radical cavity is dry and there has been no return of his symptoms.

Case 3: Male, age 17 years, had a left mastoid operation performed in Asia Minor when he was five years old. He was reoperated two years later and again when he was 11 years of age. The ear always presented a foul discharge. Three weeks prior to our seeing him he developed a postauricular swelling over the left mastoid area. This was incised and pus obtained. He presented himself for examination on Oct. 10, 1927. At that time he had a postauricular opening on the left side, through which pus drained. His middle ear was filled with cholesteatomatous masses. Functional examination of the hearing revealed total deafness on the left side. Sound was lateralized to the right ear. A radical mastoid operation was advised and refused.

The patient was seen again on Dec. 19. At that time he presented a complete left facial paralysis, which he said had been present for three days. He had no other complaint beside the discharge from the ear and the facial palsy. He was sent to the New York Polyclinic Hospital, where spinal fluid examination and blood count were normal.

A radical mastoid operation was performed on Dec. 20. A large cholesteatoma was found filling the entire mastoid cavity and middle ear. This was dissolved out with alcohol. When the middle ear was

reached, the cholesteatoma was found to involve the labyrinth in the region of the promontory. The facial nerve was free in its horizontal portion and was carefully separated away from the cholesteatomatous matrix. The wound was left open and was not packed Patient was returned to bed. On the fourth day, there was noticed a slight motion of the angle of the mouth. On the seventh day after operation, the patient was again taken to the operating room and the remaining portion of the matrix was removed from the interior of the vestibule. The entire promontory was found destroyed and cholesteatoma filled the cochlea. Nothing further was done to the labyrinth.

The following week skin was grafted into the radical cavity and the wound was closed. The flaps in the canal wall were after Panse's technique.

The patient made an uneventful recovery. The facial palsy cleared entirely and complete function returned within two months after operation. The radical cavity is dry.

Comment: In this case no evidence was available prior to operation of the extensive involvement of the labyrinth. The disease had entirely destroyed the cochlea and had invaded the vestibule. Yet at no time were there any spontaneous signs of labyrinthine irritation. The spinal fluid in this case was normal. Consequently it was thought inadvisable to extend our procedures further by exenterating the labyrinth and so disturbing the protective barriers that had been established. The result justified our judgment.

Case 4: Female, age 30 years, was seen by us on Nov. 7, 1926. She complained of a discharge from the right ear for three years. For three days prior to examination, she complained of severe right-sided headache. There was no vertigo nor falling. Otoscopic examination revealed a large marginal perforation in the right drum, through which a foul discharge came away in pulsations. Radiographic examination of the mastoid process revealed a fully developed sclerotic process with a slight irregularity in the region of the tegmen. She was admitted to the Beth Israel Hospital on Nov. 24, 1926. The blood count, spinal fluid and urine were found normal before operation was undertaken.

A radical mastoidectomy was performed on Nov. 26, 1926. A sclerotic mastoid process was found. The dura of the middle cranial fossa was found exposed by the disease and a large epidural abscess was uncovered in the region of the tegmen antri. The middle ear was filled with pus and granulations and the incus and malleus were found necrotic. The wound was left open.

Immediately after the operation, the patient complained of severe vertigo to the left. She was always found to lie on her right side. There was a marked rotary nystagmus to the left. She had marked vertigo when moved in bed and vomited repeatedly. Sound was lateralized to the right ear. The temperature rose to 103° and remained there for three days. Spinal fluid examination revealed a slight increase in intraspinal pressure, but the examination of the fluid gave normal findings. There was no change in the blood count.

Nothing was prescribed for the patient but absolute rest and daily doses of morphin. The labyrinthine symptoms gradually abated and by Dec. 15 had completed disappeared. Examination of the hearing at that time showed lateralization of sound to the left ear, that is, the unoperated side. The temperature returned to normal on the sixth day following operation.

On Dec. 18, 1926 ,the wound was closed and a Panse plastic was performed on the canal. The patient has made an uneventful recovery since. Her radical cavity is dry and the labyrinth symptoms have completely disappeared. A caloric test of both ears done on June 22, 1927, showed complete absence of all responses from the right ear, whereas the left ear yielded normal reactions. There is a complete loss of hearing in the right ear.

Comment: The patient developed a serous labyrinthitis after a radical mastoid operation. In spite of the temperature, the violent labyrinthine symptoms and the forced position, no surgery was undertaken because of the normal findings in the spinal fluid. The patient recovered and examination of the labyrinth at a later date showed a total loss of function. This is accounted for by the sudden onset of a diffuse serous labyrinthitis which destroyed the end organ by pressure necrosis.

PART II—MENINGITIS.

The treatment of otitic meningitis is one of the most important problems in otology. The futility of our therapeutic measures in the past, with the exception of scattered brilliant results from one or another means, has led most of us to accept the disease as one for which nothing can be done. The recent stimulus given to the study of this problem by Eagleton, however, has considerably promoted the interest of the profession in finding a solution of the question.

All cases of meningitis following otitic diseases are to be viewed as identical only in one respect: an inflammation of the meninges exists. In all other respects they differ. The point of entry of the infection into the subarachnoid space, the invading organism, the site of the primary meningeal involvement, the amount of protective in-

flammatory reaction, the extent to which the brain tissue itself is involved, are different in every instance of otitic meningitis. We will discuss each of these points in detail so as to arrive logically at the conclusions which we desire to present.

We accept two great pathological divisions of otitic disease, the acute and the chronic. There are two varieties of the former, the acute coalescent and the acute hemorrhagic mastoiditis. The latter presents two types also, the dangerous type and the nondangerous type, which rarely produces an intracranial disease *per se*. We must consider the meningitis that may occur in the acute hemorrhagic mastoiditis from two angles. The meningitis may be produced simultaneously with the mastoiditis by the primary blood stream infection. Viewed in this light, it is not in secondary relationship to the mastoiditis and consequently cannot be classed as an otitic meningitis. Where a meningitis occurs in this way, it is recognized clinically by the appearance of signs of meningeal irritation simultaneous with the onset of the mastoidal disease. Such a condition is fulminating and we feel that nothing can be done to combat it. It is so sudden in its attack that the body cannot combat the infection with its protective forces.

On the other hand, where meningitis occurs in the course of an acute hemorrhagic mastoiditis, it has been demonstrated definitely that the path of infection is by an extension of a thrombophlebitis of the minute mastoidal vessels into and through the dural coverings. The exact point in the anatomy at which this takes place varies and can only be definitely determined microscopically. Recorded observations seem to point to the tegmen as the point of extension in most instances.[1]

In the acute coalescent and the chronic mastoiditis of the dangerous type, the method by which the endocranium is invaded is similar. A gradual destruction of the mastoidal cells is evident, in the acute cases by destruction due to suppuration and in the chronic cases by a bone necrosis due to rarefaction subsequent to pressure. Consequently one frequently finds at the primary operation on the mastoid process a place or places on the inner table where the dural membranes have been brought into direct contact with the infectious process in the mastoid bone. It may be at the tegmen, the sinus plate, the labyrinth, Trautmann's triangle or the inner table of the suprasinal cells. In the event of a complicating meningitis, these areas of erosion in the inner table point out to us the route of invasion, and they should be made use of in the manner of which we shall speak later.

From the anatomical standpoint, the area of the first meningeal involvement is extremely important, for we feel that certain areas in the meninges are more amenable to surgical treatment than others. We feel that an infection of the pia mater in the middle cranial fossa is more responsive to treatment by surgical drainage than an infection taking place anywhere else in the subarachnoid space. This area is limited by the tentorial membrane and consequently it takes some time for a secondary infection from the aural region to spread to the large cisterns and so become diffuse. In other words, we believe that in this region a meningitis tends to remain a localized one for a long period of time before it spreads and becomes generalized. An example of this is the localized meningitis which causes the Gradenigo syndrome. This condition rarely requires any treatment other than absolute rest. Next in the order of favorable sites is the section of the posterior fossa which abuts on the posterior surfaces of the petrous portion of the temporal bone. Here we have gross anatomical barriers which tend to limit an infection of the meninges. There are the structures entering the internal auditory meatus, the ninth and tenth nerves and the root of the fifth, the cerebellar peduncles and the tentorium cerebelli. While it is true that infection in this area tends to spread rapidly to the lesser cisterns, yet the course of a meningitis originating at this site does not seem to be as virulent or as pronounced as a meningitis which starts in Trautmann's triangle, the lateral sinus or the inner table of the suprasinal cells. From an anatomical standpoint, the latter area seems to be the least favorable for preventing the progress of a meningeal infection, since direct access is had to the cisterna magna.

The amount of protective inflammatory reaction varies in each individual case. It can be stated that in the meningitis following chronic mastoiditis the protective mechanism of the body is permitted to adjust itself to the impending invasion of the meninges. In these cases, adhesions form in the vicinity of the primary site of the meningeal involvement which further tend to localize the infection in the subarachnoidal space. In the meningitis following the acute coalescent mastoiditis, however, nature is not able to throw up sufficient protective barriers in the subarachnoid space and in these instances there is not the tendency toward localization.

Where the brain tissue itself becomes involved in the inflammation by direct extension from the pia the encephalitis which follows presents as serious an aspect as the meningitis itself. The waste products which are thrown off, like choline and other lecithin groups, when in excess, cause death in themselves.

The type of infecting organism also has a part to play in the outcome of a meningeal infection. From the otological standpoint, the organisms most frequently found in purulent leptomeningitis are the streptococcus hemolyticus, the streptococcus mucosus capsulatus and the pneumococcus. We will not consider the turbecle bacillus or the influenza bacillus, the former because the very nature of the lesion contraindicates surgery and the latter because of its rarity in connection with otitic disease. The streptococcus mucosus capsulatus and the pneumococcus. We will not consider the tubercle bacillus gery. The exudate produced by these organisms is so thick and so tenacious that free drainage is impossible; and since surgery cannot offer any advantage in the treatment of meningitis but drainage, its futility can be realized in the instance of pneumococcus and mucosus meningitis.

It is to the meningitis of otitic origin produced by the streptococcus hemolyticus, therefore, that we shall limit ourselves in the discussion of the surgical treatment of meningitis. In addition, we must accept all cases of the so-called meningitis sympathica, excepting those due to brain abscess, as falling within the group amenable to surgical treatment. Meningitis sympathica is that type wherein the clinical signs of meningitis are present, the cerebrospinal fluid shows cytological, physical and chemical changes characteristic of meningitis, but no organisms are demonstrable. We concede that this is the result of a threatened invasion of the subarachnoid space and that often with the removal of the irritant the meningeal signs disappear, but we also hold that it may be considered the early stage of a purulent meningitis. It has been demonstrated at operation by Eagleton that the spinal fluid obtained on lumbar puncture may be sterile, while fluid obtained at or near the area of invasion in the region of the temporal bone shows organisms on culture. Consequently it is difficult to distinguish which case of sympathetic meningitis will get well and which case will go on to a diffuse purulent leptomeningitis. For this reason, we class all cases of sympathetic meningitis as cases of surgical meningitis, excepting only those due to a brain abscess. It will, therefore, be seen that the otitic meningitis which lends itself best to surgical treatment is that type which follows the acute coalescent or the chronic mastoiditis, wherein surgical procedures can be instituted before organisms are obtainable in the spinal fluid by lumbar puncture and wherein chemical examination of the fluid reveals a minimum amount of cholin. The most favorable cases are those wherein the primary infection of the meninges has taken place through the dura of the middle cranial fossa. Those offering the

next most favorable prognosis are the cases wherein the extension has occurred through the petrous portion of the temporal bone. The most fatal are those presenting the primary nidus in Trautmann's triangle, the sinus or the suprasinal cells.

The procedure advocated varies with the site of the invasion of the meninges. Where the middle cranial fossa is the site of the earliest meningeal involvement, we advocate a large exposure of the dura in the area of the tegmen and a wide incision into the dura. Where the labyrinth is the seat of the primary invasion, we advocate a total removal of that portion of the petrosa as far down as the internal auditory meatus, removal of the entire inner table over Trautmann's triangle and a wide incision into the dura thus exposed. Where the sinus is the site, or where the infection has gained entrance through Trautmann's triangle, a drainage of the cisterna magna is advised. Operation should be undertaken as early as possible. If one waits until the purulent meningitis is evident on lumbar puncture, no operative procedure will be successful.

We must differentiate the meningitis following otogenic disease from the meningitis following trauma and operative measures in the cranial cavity. In the instance of a meningitis which develops subsequently to a lumbar puncture or an operation for the removal of an intracranial tumor, the infection takes place as a result of the entrance of bacteria into the subarachnoid space at the point of trauma and their propagation in the fluid. The meninges are secondarily affected in these instances. Consequently, early drainage of the cerebrospinal fluid system by a cisterna magna operation will evacuate the infected fluid before the dural linings are infected. That is the reason for the successes reported by Dandy. On the other hand, where a meningitis develops following an otitic disease, it is the meninges which are first infected and the fluid is infected secondarily. That is the reason why drainage instituted in an area where the meninges are not affected will do little to relieve the condition. In otitic meningitis drainage must be instituted in the area of infection to afford any hope of success.

We present herewith three cases of meningitis: in two the infection was definitely traced to the labyrinth as the source; in the third, it was felt that the lesion gained entrance through an area of erosion in Trautmann's triangle. In this last case a cisterna magna drainage was performed for the relief of the meningitis, whereas in the other two cases the lesser cisterns were drained through the labyrinth and Trautmann's triangle. It is true that in none of these cases were organisms isolated from the spinal fluid at any time. Nevertheless,

we feel that if we had not operated early, a suppurative meningitis would have ensued and the patient died. We base this opinion on observations previously made wherein spinal fluid findings like the ones noted in these three cases preceded by some days the discovery of organisms and pus in the spinal fluid and subsequent death.

Case 1: Male, age 21 years, was admitted to the New York Polyclinic Hospital on April 20, 1928. Eight weeks prior to this he suffered from an acute purulent otitis media in the left ear. This was treated by local applications of various antiseptics, douching and suction. At no time did the patient complain of pain in the ear. For the week immediately preceding admission to the hospital, the patient was very dizzy, was not able to walk without staggering and was nauseous most of the time. The discharge from the ear was profuse There was no fever at any time.

Examination of the ear revealed a profuse purulent discharge, which came away under pressure. Compression of the internal jugular vein on the left side caused the pus to gush out through the large perforation in the drum. The mastoid process was not tender to the touch. Roentgen examination revealed a total coalescence of the cellular elements in the left mastoid process. Examination of the hearing showed a total loss of air conduction for the tuning forks. The Weber test revealed lateralization to the right ear. There was present a mixed rotary and horizontal nystagmus, with the quick component to the left. Fistula test was negative. Caloric test showed complete absence of function in the left vestibular apparatus.

Examination of the blood showed the following: On the day of admission the red cells numbered 4,500,000; the hemoglobin was 78 per cent; the total white count was 12,000, and the polynuclear percentage was 82. The blood culture taken on that day proved to be sterile after 72 hours' incubation. Examination of the spinal fluid showed normal findings.

On April 20, 1928, a radical mastoid operation was performed on the left ear as the first step in the treatment of this patient. The entire mastoid process was found destroyed and the cells were completely coalesced. An erosion of the promontory was found and another area of necrosis was evident in the region of the superior semicircular canal. The incus and malleus were removed intact. The wound was left open and lightly packed with iodoform gauze.

On the day following operation, the patient complained of slight headache over both eyes. Examination of the eye grounds revealed normal fundi. There was no change in the blood count. Physical examination showed a beginning rigidity of the neck, a positive Ker-

nig on the left side and an absence of upper abdominal superficial reflexes. Examination of the spinal fluid showed no abnormalities. The patient was kept absolutely at rest and was given a saturated solution of magnesium sulphate by rectum twice daily.

The condition of the man remained unchanged for the next two days. On the fourth day after operation, spinal tap showed the fluid to be under increased pressure and cloudy; there were 680 cells to the c.c., of which 92 per cent were polynuciears; albumin and globulin were present; copper reduction was present; there were no organisms on culture.

With this finding, we immediately advised drainage of the posterior fossa in the region of the lesser cisterns. The patient was accordingly operated on April 26, 1928. The radical mastoid wound was opened and the horizontal portion of the facial nerve was freed from its bony canal and pushed downward. The inner table over Trautmann's triangle was then removed and the petrous portion of the temporal bone housing the posterior and horizontal semicircular canals was removed by chisel and gouge until the dura of the posterior fossa was reached in the vicinity of the posterior surface of the petrous bone. The dura was carefully separated from the petrous bone and a wide incision was made in the dura. Cerebrospinal fluid escaped under great pressure. A culture of this fluid was sterile. The facial was then lifted upward, the promontory was removed and the two windows were joined so as to drain the vestibule. The wound was not packed, in order to permit free drainage of the cerebrospinal fluid.

The day following this second operation found the patient comfortable. All headache was relieved. A distinct facial paralysis was present on the left side. All clinical signs of meningeal irritation had disappeared. The spinal fluid on this day showed a reduction in the number of cells to 340 to the c.c. Albumin and globulin were still present; the sugar content was normal; culture was sterile.

On the fifth day following the second operation, a normal spinal fluid was obtained, with normal findings on chemical and cytological examinations. A degree of function remained in the facial nerve as manifested by the reaction of degeneration. Stimulation of the nerve was given in the form of massage and infra-red lamp.

On May 2, 1928, the plastic operation on the posterior wall of the external auditory canal was performed and flaps were cut after Panse's technique. The posterior wound was closed. The patient made an uneventful recovery thereafter. The vertigo persisted for five months and has now disappeared. The cavity is completely

epidermatized. The facial palsy has completely cleared up except for a slight loss of motion in the region of the forehead.

Comment: In this case the finding of labyrinthine irritation on clinical examination, coupled with the evidences of erosion of the outer labyrinthine capsule at the time of operation, indicated the route of infection to be through the labyrinth into the posterior fossa bordering the petrous pyramid. The lesser cisterns were drained and the patient recovered.

Case 2: Girl, age 17 years, had been suffering from a chronic right aural discharge for 13 years. When first examined on Feb. 3, 1928, she complained of severe pain in the right ear, excessive vertigo and falling in all directions, and severe occipital headache. These symptoms had come on one week previously, concomitantly with a cessation of the aural discharge.

Examination of the right ear revealed a marginal perforation, through which a scant, foul discharge was evident. A rotary nystagmus was present in all directions. The Romberg was positive. Examination of the hearing showed only a slight deafness in the right ear. The caloric tests showed normal vestibular function. The fistula test was negative. The patient was admitted to the Polyclinic Hospital and a blood count and spinal fluid examination were done. These revealed normal findings. The blood count showed 4,600,000 red cells, 80 per cent hemoglobin, 9,000 white cells and 69 per cent polynuclears.

A simple mastoid operation was performed and the procedure extended forward to remove the outer attic wall. The incus and malleus were found intact and, in view of the slight loss in hearing, were left *in situ*. The mastoid process was completely eburnated and merely a small area of necrosis was found in the region of the antrum and in Trautmann's triangle.

All the patient's symptoms disappeared immediately after operation. She was permitted to be out of bed on the sixth day after the mastoidectomy. She had no fever at any time. She was discharged from the hospital on March 13, 1928.

Four days after discharge from the hospital she returned with the same symptoms as she had had prior to operation. The vertigo and nystagmus were pronounced. The staggering gait was prominent. She had severe occipital headache, which was not relieved by any narcotic. Examination showed the wound to be in excellent condition. The middle ear was dry. Re-examination of the labyrinth showed no abnormalities in the responses to the cold caloric. There was some rigidity of the neck, but this was not found at examination

12 hours later. No Kernig nor Babinski. A cerebellar abscess was strongly suspected and Dr. Sharpe was called in consultation. He felt that the lesion was in the labyrinth and accordingly she was re-operated on the mastoid process and the wound was thoroughly inspected. No evidence of any necrosis was found in the outer labyrinthine capsule. The sinus and the dura in the middle and posterior fossae were inspected and found normal in appearance.

Five days later examination of the spinal fluid showed an increased pressure, albumin and globulin, 760 cells to the c.c., sugar present and no organisms on culture. Believing the case to be one of an early meningitis, due possibly to an intracerebellar abscess or to an extension of the disease through the necrotic area in the region of Trautmann's triangle, it was decided to perform a cisternal drainage.

A large exposure of both cerebellar hemispheres was made and no bulging of either hemisphere was noted. This, in Dr. Sharpe's opinion, ruled out an intracerebellar suppuration. The exposure of the pial covering of the cisterna magna in the region of the fourth ventricle, however, showed it to be covered with numerous patches of inflammatory exudate. This was evident throughout the area of the posterior fossa exposed by the operative procedure. The cisterna was drained and the fluid came out under great pressure. Culture of this fluid proved sterile.

Subsequent to operation, the patient made a gradual improvement. At no time was the temperature any higher than 101°. Her vertigo and nystagmus gradually abated and within five weeks both the mastoid wound and the occipital wound had healed. Successive lumbar puncture showed an entire disappearance of the evidences of meningeal reaction. The patient is now entirely recovered.

Comment: The finding of a necrotic area in the region of Trautmann's triangle, coupled with symptoms of cerebellar inco-ordination, pointed out the route of infection to be through Trautmann's triangle directly into the cisterna magna. Consequently a cisterna magna operation was the procedure selected in order to drain at the site of the infected area.

Case 3: Male, age 7 years, was admitted to the United Israel-Zion Hospital on March 5, 1927. Eighteen days prior to admission, the child complained of pain in the right ear. A paracentesis was performed two days later. The child then developed a septic temperature and complained of severe frontal headaches.

On admission to the hospital there was found a pulsating discharge from the right ear, which came out under pressure. There was some tenderness over the right mastoid and considerable edema of the soft

tissues. The cervical glands on the right side were all enlarged. The temperature on admission was 103.6°. The blood count showed 4,480,000 red blood cells; hemoglobin, 85 per cent; 23,800 white blood cells; polys., 88 per cent. The urine was negative.

On March 6 a simple mastoid operation was performed. An acute hemorrhagic mastoiditis was found. The sinus and dura were uncovered for inspection and found normal. The mastoid was exenterated down to the inner table throughout and the wound was then drained with rubber dam.

The temperature fell by lysis and returned to normal on the seventh day after operation. The patient was discharged from the hospital on the ninth day. At the time of discharge the blood count showed normal findings. The wound had completely healed within three weeks after operation and the child had returned to school, feeling perfectly well.

On April 21 patient began again to complain of severe frontal headache and a swelling was noticed over the old scar behind the right ear. He was readmitted to the hospital on that day. Examination revealed a positive Kernig on both sides and a marked rigidity of the neck. The spinal fluid came out under marked pressure, but was negative on serological and chemical examination. The total cell count was three per c.c. The temperature was 102°.

The child was given large doses of magnesium sulphate by rectum twice daily. Under this treatment all signs of meningeal irritation disappeared within three days. The swelling behind the mastoid subsided under wet dressings and was not evident after the second day in the hospital. The temperature reached 99.8° on the fourth day and remained there. The red cell and hemoglobin estimations were at all times normal and the total white count dropped from 27,600, with 84 per cent polys on the day of admission, to 13,900, with 74 per cent polys on March 29.

However, the child continued to complain of severe headache over the frontal area. Radiographic examination of the nasal sinuses revealed no pathology. The temperature continued below 100°. On May 6 a weakness of the right side of the face was noted. The eye grounds on that day were found normal.

On May 11 examination by Dr. E. D. Friedman showed the facial paralysis on the right side to be complete, with some involvement of taste on the right side of the tongue. The right corneal reflex was diminished. The rest of the neurological status was negative. Spinal fluid examinations on four different occasions were normal.

On May 16 there was noted a return of the Kernig and a slight rigidity of the neck. There also appeared to be a slight spontaneous nystagmus in the horizontal plane when the patient looked to the left. The spinal tap on that date showed a slight increase in pressure, but no other abnormalities. The temperature began to rise and for the next three days reached 101° daily. On May 21 the temperature suddenly rose to 104°. A spinal tap revealed cloudy fluid under increased pressure. There were 280 cells to the c.c., with 90 per cent polys; albumin and globulin were present and sugar was reduced. No bacterial growth was evident on culture.

The patient was taken to the operating room on May 22 and the mastoid wound was reopened. No pus was found anywhere. The wound was converted into a radical cavity and a complete exenteration of the labyrinth was performed because of the nystagmoid movements and the involvement of the facial and trigeminal nerves. These findings indicated the seat of the lesion to be in the posterior fossa in the region of the petrous pyramid. The entire dura of Trautmann's triangle was then exposed and the petrous bone removed as far as the internal auditory meatus. The dura of the posterior fossa was then incised and the cerebrospinal fluid allowed to escape. No packing was inserted. The spinal fluid from this area was found to be sterile on culture.

The child's temperature then returned to normal and remained so until the time of discharge on June 12, 1927. All signs of meningeal irritation gradually cleared up and were entirely gone on June 2. The facial weakness became less and less noticeable and eventually disappeared. The radical was completed on June 3 by sewing the wound and performing the plastic operation on the posterior canal wall. The patient has made a complete recovery.

Spinal fluid examinations subsequent to the operation for cisterna minor drainage were as follows: May 26: 180 cells, 60 per cent polys, otherwise normal. May 31: 60 cells, 54 per cent polys, otherwise normal. June 5: 5 cells, 100 per cent lymphs, otherwise normal.

Comment: This case presents an acute hemorrhagic mastoiditis, with subsequent involvement of the dural lining of the posterior fossa. The route of infection to the subarachnoid space was shown by the involvement of the facial and the trigeminal and by the transitory nystagmus. No organisms were found, yet the child presented all the clinical signs of a meningitis. All symptoms cleared up and the spinal fluid returned to normal after drainage of the cisterna minor on the affected side.

CONCLUSIONS.

1. Where a circumscribed purulent labyrinthitis exists, regardless of its extent, no surgery on the labyrinth is indicated when the spinal fluid shows normal chemical, cytological and bacteriological findings.

2. Where a diffuse serous labyrinthitis exists which is the direct result of a pathological process in the middle ear or mastoid, no surgery on the labyrinth is required.

3. In both the diffuse serous and the circumscribed purulent labyrinthitis, removal of the causative factor is sufficient to result in a cure of the lesions.

4. A diffuse purulent labyrinthitis requires a labyrinthectomy only when the cerebrospinal fluid presents evidence of a threatened invasion of the subarachnoid space. In such cases, we feel that surgery on the meninges is also indicated.

5. Otitic meningitis is amenable to surgical treatment in the very early stages of the disease. Surgical measures in these cases must be planned with attention to the type of organism found, the route of infection and the area of the subarachnoid space first involved.

6. The route of infection is an important guide to the prognosis. Where the route leads to the middle cranial fossa, the prognosis is most favorable. Next is the route of infection leading into the lesser cisterns. The infection which takes place through Trautmann's triangle or through the lateral sinus gives the worst prognostic outlook.

When doing the original surgery on the mastoid process, the route of infection should always be noted, in view of the possibility of subsequent endocranial complications. Should such complications supervene, one can utilize the data obtained at the primary operation; for the surgical attack should be made directly on the area of the subarachnoid space first infected.

7. A purulent meningitis in its earlier stages may reveal no organism in the spinal fluid. Consequently, we disadvise waiting for the relief of this condition. Operative measures should be instituted very early; and the indication for them should be the clinical syndrome of meningitis, associated with a purulent spinal fluid.

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NEURO-SURGICAL TREATMENT OF BRAIN ABSCESS.*

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Brain abscess occurs most frequently, according to location, in the following order: Temporo-sphenoidal, cerebellar and frontal. The most favorable case for operation, and one in which there are no unfavorable sequelae, such as headaches, dizziness, mental deterioration, Jacksonian epilepsy and so forth, is the temporo-sphenoidal lobe abscess, and especially if it is encapsulated. The next most favorable is the frontal lobe abscess, and the least is the cerebellar.

In dealing with intracranial complications, especially associated with sinus and mastoid infection, one should be careful to differentiate between an epidural abscess and a brain abscess. This differentiation is not always readily done, especially in the early stages of the infection. Intracranial explorations have been performed with negative results, due to the fact that the lesion is an extradural one. When in doubt it is advised that the probable source of infection, for example, mastoid, frontal sinus, etc., should first be operated upon.

Although various forms of drainage materials have been advised and used in operations for brain abscess, none of them adequately drains in the true sense. The most logical drain is the conical wire mesh drain (Mosher). When this drain is used and the patient recovers, it is believed to be due to the fact that the abscess cavity herniates against the brain in such a manner that the residual portion of the abscess cavity "plasters" itself against the drain and then herniates through the opening in which the drain was inserted, thus effecting the cure. However, it is believed that not all of these abscess cavities will symmetrically collapse about this cone-shaped drain, so that in such cases secondary pockets, or abscesses, will surely develop.

In this paper, as in previous ones, it is considered advisable to discard drains, as such, entirely, and instead to allow the abscess cavity completely to herniate itself from the intracranial space through a bony opening, made sufficiently large and so placed as to allow complete herniation to occur. By so doing there is nothing to be drained, due to the fact that the abscess cavity has completely everted itself and has become the dome of the hernia cerebri, infec-

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tion to the meninges is unlikely, the increased intracranial pressure which produces a slow pulse and coma is immediately lessened, and there is no danger of the formation of a secondary abscess or rupture of the abscess into the ventricle. One or more of these conditions enumerated above, if not prevented, produce a fatal result. As described in previous papers, it is here advised that:

1. The exact localization of the abscess be made by the use of a brain cannula through a trephine opening.

2. The trephine opening should then be enlarged so that it corresponds in position, size and location with the abscess cavity, *i. e.*, the opening should be so enlarged that it is directly over the abscess.

3. Complete herniation of the abscess cavity so that no drains are required.

4. Dakinization of the area until the hernia cerebri has subsided.

5. Epithelization of the area.

In dealing with brain abscess one should consider the case as one in which there is the faintest possibility of saving life and completely disregard statistics, *i. e.*, one must take the bad with the good and operate upon any type of abscess in which there is the slightest chance of recovery. Those cases, in which there is a concomitant, severe meningitis, are poor risks. It is believed that a higher percentage of recovery would follow a delayed operation than one which is immediately done during the severe meningitis. All cases of encapsulated brain abscess evidently went through the period of more or less severe meningitis and were able to weather the storm until encapsulation could take place.

Six cases operated upon by this method have recovered, the first one having been done in 1920. Several other unfavorable cases have died. Two cases died due to the late operation on the frontal sinus, which operation should have been performed sooner. Two additional cases are reported: One of a frontal lobe abscess, and the other temporo-sphenoidal lobe abscess.

A plea is made for more hearty and thorough co-operation between the neurologist, otologist and the neuro-surgeon.

114 East 54th Street.

AIDS FOR THE HARD OF HEARING.*

DR. HORACE NEWHART, Minneapolis, Minn.

The important service now rendered the hard of hearing by many recently improved aids, and the relatively meager amount of literature on the subject now available, justify a paper of the above title. Fairness to the various manufacturers obviously precludes the specific mention of names. The most important articles dealing with the subject have been published during the past year as the direct result of the interest and activities of the deafened themselves, represented by their national organization, the American Federation of Organizations for the Hard of Hearing. The articles worthy of special mention include that portion of the report of the Committee on Scientific Research relating to "A Method for Rating the Performance of Hearing Aids", of which Dr. Harvey Fletcher is Chairman, and the Report of the Committee to "Survey Instrumental Aids to Hearing", Dr. Douglas Macfarlan, Chairman. Both reports are published in the Proceedings of the American Federation of Organizations for the Hard of Hearing for 1927. The work of the latter committee was carried on with the generous co-operation of the United States Bureau of Standards. A further recent contribution by Dr. Fletcher is entitled, "Instrumental Problems of the Deaf", presented before the conference on the Problems of the Deaf called by Dr. Knight Dunlap, Chairman of the Section on Anthropology and Psychology of the National Research Council, Washington, D. C., Jan. 20 and 21, 1928.

Mechanical aids to hearing are divided into two groups, the non-electric and the electric.

To the former class belond the many varied types of horns, conches, speaking tubes, auricles, etc., of different materials with which the past generation was more especially familiar. They depend for their effectiveness on the collection and amplification through resonance of the sounds of speech, so that they are conveyed to the ear in intensified form with the least possible distortion. Some instruments of this type are used to transmit the sounds by bone conduction through the teeth or cranial bones. Many individuals with a moderate hearing loss find this type serviceable and inexpensive. The chief objection is founded on their conspicuousness, weight

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and unwieldiness. According to Miss Annetta W. Peck, of the New York League for the Hard of Hearing, whose experience with aids to the hard of hearing is most extensive, many more people should be encouraged to use the nonelectric aids earlier and more persistently than is now the case.

Electric aids to hearing have been extensively used only during the past 25 years. It should be recalled in passing, that it was due to the efforts of Professor Alexander Graham Bell to produce an electric aid for his hard of hearing fiancee, later his wife, that the world today enjoys the telephone.

For practical purposes, in connection with the use of electric hearing aids, Fletcher groups the hard of hearing into four classes: *1*. Those who readily hear the ordinary conversation voice at 2 to 4 feet and who show with the audiometer a loss of less than 30 sensation units in the better ear throughout the range of the human voice, *i. e.*, from 100 to 3,000 double vibrations per second. *2*. Those whose loss is from 30 to 60 sensation units for the same pitch range. *3*. Those with a loss of from 60 to 80 sensation units. *4*. Those whose loss is greater than 80 sensation units. He states that if electric amplification could be made "ideally free from distortions", the needs of the first three groups could be met by two instruments, the first sufficiently powerful to overcome a hearing loss of 30 sensation units, equivalent to a magnification of 1,000 times, the second having a power sufficient to overcome a loss of 50 sensation units, representing a magnification of 100,000 times. The first group require no special air for interpreting conversation at close range, and in an ordinary room with the usual amount of noise, such a person should hear up to a distance of 10 feet with the aid of the weaker instrument, for a person speaking in such an environment instinctively increases the intensity of his voice to overcome the handicap produced for normal individuals by the noises present. In a quiet place, however, a person of the first group would need an instrument of the weaker type for distant conversation, that is with the speaker further away than 10 feet. A person belonging to group two would require the less powerful instruments for close work. For distant hearing, it would be necessary to place the transmitter near the speaker or to resort to the more powerful instrument. The requirements of a person in group three would be met only by the more powerful instrument. In practice these various requirements are largely met by giving increased intensity, by adding more transmitters and otherwise increasing the volume of output through the employment of resonating bodies. All types of instruments are fitted with controlling

devices, permitting the selection of all intensities below the maximum at the will of the user. Added power is secured in some types by substituting for the "midget" receiver a larger one used with the headband.

Persons of group four present peculiar problems and require instruments of special construction, with the disadvantage of increased distortion and lessened portability. Nevertheless, instruments of this type have been recently perfected with vacuum tubes and improved transmitters, and are more efficient in volume and relative freedom from distortion. The larger models are not portable. Equipped with multiple outlets having individual control and fitted with phonograph attachment, they are most useful aids to the teacher in schools for the deaf, helping to get over to those with but a small remnant of hearing, rhythm, accent and pitch.

The nonportable type is also used most successfully in the home or place of business. With its aid, the ordinary telephone is easily used. It should be stated that our telephone companies are now prepared to supply, on a rental basis, special equipment for amplifying the sounds of the receiver so that those with a considerable hearing loss can now use the telephone with satisfaction.

In a practical way, we may properly ask what should be the duty and attitude of the otologist with respect to aids for the hard of hearing? He should obviously first of all inform himself regarding the possibilities and limitations of the means now available to help the deafened. He may no longer be able to improve his patient's hearing by treatment. Too often, he cannot even hope to successfully conserve what hearing remains. Nevertheless, the patient rightly looks to the medical man as the one person peculiarly able to understand his special needs. He expects of the latter, sympathy, guidance and such advice as will help him most effectively to keep in touch with the world, which he feels is gradually receding from him.

In performing this function, the physician should urgently advise two things, which though of the greatest importance, are often neglected or postponed, to the great disadvantage of the patient. These are, first, to encourage the person who has a hearing loss sufficient to be a handicap, with the prospect of further loss, to at once procure the best hearing device he can afford; and, second, to begin seriously the study of lip-reading, or speech-reading, the greatest of all boons to the deafened.

The tendency to regard these measures as something to be employed only as a last resort, is to be vigorously condemned as fallacious and pernicious. If a person delays adopting these aids until

he is severely deafened, he loses much valuable time, during which he unnecessarily burdens himself with added exhausting effort, and afflicts his friends. If he resorts to the instrumental aid late, it requires a far greater effort to re-educate his ears. He finds special difficulty in excluding or ignoring all the adventitious sounds which the normal person from lifelong habit unconsciously suppresses. The adventitious sounds are all necessarily amplified equally with the useful sounds of speech, and constitute the greatest obstacle for one beginning the use of an electric aid. Therefore, it is most important to encourage every beginner to persist in his attempts to use the instrument over a considerable period, until he acquires sufficient familiarity and skill to make the advantages outweigh the disadvantages. Then he finds real happiness in his new accomplishment.

The great advantage of proficiency in lip-reading as a supplement to an instrumental aid is not adequately appreciated. Even persons with normal hearing rely unconsciously upon lip-reading in their interpretation of ordinary speech. To the hard of hearing, the need of this supplementary aid is emphatically greater. In fact, it is indispensable. For those with a severe hearing loss, even with the best hearing aid, many of the consonants and diphthongs are not perceived, though the vowels are still heard. Lip-reading enables such persons to supply the unheard elements of speech. Accordingly, the otologist should insist on the early study of lip-reading as soon as he discovers in his patient a permanent or threatened handicap.

Instruction in lip-reading is now advocated for such school children as are found on audiometric test to have a hearing defect sufficient to be a possible factor in causing retardation in their work. The repeater in school is abhorred alike by the educator and the taxpayer. Some of our cities go so far as to provide through the public schools instruction in lip-reading for the adult hard of hearing.

Of very great assistance to the hard of hearing, and for that matter to the rest of us who are not so afflicted, would be the promotion of a national movement to encourage the proper enunciation and pronunciation of the English language. This point is so obvious as to require no further elucidation, though in our daily contacts we are apt to lose sight of its importance.

Previous to the efforts of the American Federation of Organizations for the Hard of Hearing to investigate hearing aids, one occasionally heard various criticisms and even complaints regarding the instruments or the methods employed in selling them. These related, in some instances, to extravagant or unwarranted claims by some manufacturers or distributors for the performance of their products

or to their methods of selling them. Other complaints were in the nature of protests against the expense of maintenance, largely because of the employment of special types of batteries. Many felt that the charges for a trial rental were unjust.

These causes for criticism so far as they existed have largely been corrected, especially by the larger manufacturers, who are now rendering a highly ethical and efficient service. The mechanical features of many instruments have lately been greatly improved. The newer midget receivers are remarkable in lightness, compactness and inconspicuousness, the latter quality making an especial appeal to the many who seem never to overcome their sensitiveness to their handicap. Some manufacturers employ standard battery units, such as are ordinarily used in flashlights, which are inexpensive and are everywhere procurable.

The otologist is often asked regarding the cost of an electric hearing aid. Some makes put out a simple, efficient outfit for $25, which is useful, however, only for those with a relatively small hearing loss. In general, the cost is proportionate to the amplification. Several makers produce excellent instruments, with a wide range of power, and with various accessories in the form of different receivers and containers for the amplifiers, which serve as resonators, for from $65 to $100. Instruments for the very hard of hearing employing vacuum tubes are proportionately more expensive, the nonportable types for home and office use costing several hundred dollars. Those for school use are even more expensive.

A greatly appreciated service now provided by several local organizations for the hard of hearing is the maintenance in their quarters of a permanent exhibit of various types of hearing aids, which may be inspected and tried out by prospective users. Those in charge have no commercial connection with the manufacturers. In some cases, the social worker endeavors to procure for the needy, discarded or abandoned hearing aids at a minimum cost, or gratuitously.

Since different persons have very different requirements, which can be best met only by one of many hearing aids, it is evident that the selection of a device which will give the greatest satisfaction is a very personal matter. Final purchase should be made only after a thorough trial, for which the manufacturers make liberal provisions. Determination of the type to be tried out will be greatly facilitated by a careful otologic examination, which in our experience is made more valuable by the audiometer test. An audiogram is very useful if the patient cannot go personally to the distributor, and is helpful in watching the general progress of the case.

Statements have been made, leading to the inference that the use of electric hearing aids has a curative effect. Every otologist should vigorously combat such claims, whether made directly or by implication, for they are calculated to abuse the credulity and ignorance of the afflicted. To the physician it must be clear that no hearing device can remove the pathologic causes of deafness. We admit that in many cases there is an improvement in the ability to hear speech, following the use of a hearing aid. This, however, is the result of the acquisition of greater skill in the interpretation of speech sounds. Actual increase in acuity for pure tones as the result of using a hearing device, so far as we know, has not been scientifically demonstrated.

On the other hand, subjecting the auditory apparatus, whether it is normal or has suffered material impairment, to loud sounds for a prolonged period in the hope of improving the hearing, is to be condemned as harmful. Hence the danger of too great amplification.

It should be emphatically stated that the otologist's duty to his hard of hearing patient does not cease when he has prescribed a hearing aid and lip-reading. Too often, however, the patient is permitted to gain the impression that he no longer needs otologic care. On the contrary, so long as he has remaining a useful remnant of hearing acuity, he should at least be under the observation of an interested medical man, so that no possible factor may be overlooked which might lead to greater hearing loss. The deafened person, more than all others, needs to conserve his hearing. He should have a careful examination, including an audiometric test, at stated intervals. He should at all times be guarded from the deleterious influences of excesses in food, tobacco and alcohol. He should avoid the harmful effects of improper elimination, inflammatory disturbances of the upper respiratory tract, including acute and chronic sinus disease, focal infections, wherever located, and severe fatigue. Such a program can be best directed only by the otologist, to the end that the patient may retain the highest possible hearing efficiency.

Among the most important problems which confront those who would effectively help the hard of hearing individual is the matter of his psychology. It is perhaps the greatest obstacle to his success and enjoyment of life. The average deafened person by virtue of his impairment has lost confidence in himself. He soon develops an inferiority complex, which grows upon him through the isolation with which he seeks to conceal his handicap or to avoid being constantly reminded of it. This situation is being met most efficiently through the activities of the local organizations for the hard of hearing which

now exist in many of our cities. No one understands better than the deafened themselves the needs of one with acquired deafness, and the otologist who will induce his hard of hearing patient to associate himself early with a group of this kind is rendering a distinctive service. The achievements in the way of rehabilitation of the hard of hearing person through these agencies are in many instances most surprising.

Thus safeguarded, and fortified with the mastery of a properly selected instrumental aid, having acquired his maximum of skill in lip-reading, the average hard of hearing person has conquered to a great extent his handicap, and should courageously take his share of the responsibilities and joys of life.

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DISCUSSION.

DR. MAX A. GOLDSTEIN, St. Louis, Mo.: The few remarks that I shall make on this very timely paper are based in substance on one statement in the paper: "No hearing device can remove the pathologic causes of deafness." I was standing in the back of the room with one of our St. Louis colleagues and I said, "What a wonderful thing radio amplification is for a large group like this," and he replied, "Yes, but to a certain extent it changes the quality of

the voice, and to a certain extent impairs the personal equation of the speaker." That is true about every electric hearing device. The two hearing devices which are of use to the deafened individual are the electric and the non-electric. All the electric devices are based on the radio and the telephone principles, and there is no doubt that the deafened person who uses an electric device is confronted with the fact that the sound wave goes through a metal diaphragm and is more the fact that the sound wave is transmitted through an electrically activated metal diaphragm and is more or less distorted. I know of one gentleman who has been an otosclerotic for years and has used various types of electric devices, and finally abandoned all of them, coming back to the old-fashioned wire spiral, silk covered hearing tube, and he gets more satisfaction out of that than any of the amplified radio machines.

Another factor about the electric device is, that at a distance of more than a few feet, the amplification of the voice is not satisfactory. You must be pretty close to the amplifier in order to get thorough satisfaction in the reception of sound.

I am very glad that Dr. Newhart has emphasized as perhaps the most important aid to hearing the use of lip-reading. Lip-reading has come into its own as a universal substitute for the adult with defective hearing. About the child, of course, there is more to be said. The adult who loses his hearing after he has thoroughly mastered automatic speech is one equation; the child who has never been taught to hear and never has had an opportunity to hear requires not only lip-reading as a means of contact from you to him, but he requires in addition the ability to talk, which is his contact with you. But when an adult should use lip-reading should be settled by the otologist, and every otologist should be in position to advise his hard of hearing patients to master lip-reading as about the only satisfactory and permanent aid to his handicap.

I appeal to you all—this is a large body, that can do a great deal of good in the propagation of a thing that is thoroughly satisfactory and reliable—I appeal to you all to regard lip-reading as an asset to the deafened man, a thing to have in your minds every time you advise a deafened person that you can do no more by surgery or therapy.

Dr. Robert Sonnenschein, Chicago, Ill.: This excellent presentation, concise and complete, leaves very little for discussion. I would like merely to emphasize three or four points.

With reference to mechanical aids, one should remember that Dr. Paul Sabine, of the Riverbank Laboratory, showed some years ago, that with the exception of the largest (hence, most cumbersome and inconvenient) types of tubes or horns, the hand placed against the edge of the auricle, with the concavity forwards, acts just as well as a collector of sound waves and as a resonator. Years ago, I happened to obtain a few tubes made of copper, which were very light, easily carried and gave tremendous aid to hearing. They were of English manufacture, rather expensive and difficult to get, but they were the best type of hearing tubes I have ever seen.

As regard the electric hearing devices, we all realize several defects, or disadvantages. There is the rather high price, some costing $65.00, the difficulty with batteries, the great weight of some of the appliances, and the adventitious sounds produced by so many of them. These forms of apparatus give best results in middle ear, or better said, conduction apparatus impairment. C. von Eicken (Berlin), at the meeting of the German Otolaryngological

Society, June, 1927, presented his findings with reference to the types of impaired hearing favorably or otherwise influenced by the electric devices. He states that those most helped were cases of advanced middle ear impairment, less those cases of combined middle and inner ear affection, and the least benefit was derived in cases of auditory nerve impairment. With this statement, I am quite well in agreement.

The matter of choice of a particular type of apparatus (provided any is advisable) is very important. Some patients respond better to one kind of hearing device (whether it be mechanical or electric) than others. It is therefore necessary, in my opinion, that the patient be permitted by the dealers not only to test out the appliance at the shop, where they may be excited or disturbed by noises, the presence of other persons, etc., and can thus not make a good choice, or perhaps even decide whether the apparatus in question aids them at all, but that they should be allowed to take the apparatus home on approval for several days or weeks in order to be able to definitely determine the value of the instrument to them. I am glad Dr. Newhart finds the dealers now more willing to do this, for my experience, thus far, has been rather disagreeable. After telling the patients to buy no apparatus unless they were allowed to try it out at home, the answer usually has been that the dealer refuses such a privilege. Perhaps the publicity given this matter by the Committee, Drs. Newhart, McFarlan and Walker, has caused at least some of the dealers to see the "light".

Lastly, I wish to endorse and emphasize what the essayist has said about lip-reading. Acquisition of this art proves a wonderful aid to the hard of hearing, not merely in their ability to mix with their fellows in business, profession or society, but it relieves them of the constant exacting and exhausting efforts of straining in order to hear the spoken word.

The trio of, first, a careful examination of hearing in order to make an accurate diagnosis, to pronounce a prognosis, and decide which type of hearing device may help; second, selecting such apparatus; and third, having the individual, as soon as possible and as intensively as he can, study lip-reading—this trio of endeavor will mitigate in very considerable measure the distressing disabilities of the unfortunate hard of hearing.

Dr. Sam E. Roberts, Kansas City, Mo.: There is no question about the efficiency of artificial aids to hearing. The chief difficulty we have experienced is getting the patient to wear the apparatus after it has been provided, due to a false pride. They do not want to wear this sign of a physical defect. I have assisted many patients to select a device, demonstrated its usefulness by tests beyond question of doubt and seemingly to the patient's satisfaction, only to find after a few weeks that they have discontinued its use.

Dr. Newhart stated that patients should be introduced to artificial aids for hearing first. Perhaps he made that statement because his subject was artificial aids to hearing. Personally, I think they should be first introduced to lip-reading. Many persons not over 30 or 40 acquire lip-reading readily, and become more proficient the longer they use it. In selecting the apparatus, it should be fitted to the patient's needs, and not the patient's needs to any kind of device.

Dr. John F. Barnhill, Indianapolis, Ind.: This subject has heretofore received far too little attention. However, as one notes that a large percentage of members in attendance have remained to hear Dr. Newhart's paper, it

becomes evident that there is a rapidly-growing interest among otologists in the management of the hard of hearing.

What that is worth while may be done for these unfortunates? Some of them may be improved by treating the ears, some may have the progress of their deafness arrested, and many are not improved at all by treatment. All those who may be benefited in any way by physical treatment should be advised to receive such measures so long as noticeable benefit follows. There are also many among the class who are not benefited who should, for psychologic reasons, receive occasional treatment, for to abandon them as hopeless often does great harm in leading them to take up with quackery methods and promises of the worst order. For the otologist to see such cases occasionally affords an opportunity of noting the progress of the affection and of providing some aid to hearing at the proper time and of the most suitable type. Merely to send the hard of hearing individual to a store, with the suggestion that he buy some specified aid to hearing, is not adequate. The otologist should not only prescribe the type of aid that is most indicated, but he should also see the individual afterward sufficiently often to note the effect and to adjust the instrument to the best advantage. Not infrequently, the patient refuses to wear a device because insufficient and wearisome, when if he is encouraged and the aid properly adjusted, the result is highly satisfying. Best results are often not obtained from electric devices should the patient try to wear the aid constantly, for the reason that all sounds and all voices are conducted in a new, strange, and often tiresome way, the patient becoming excessively fatigued mentally, and greatly disturbed physically. A few hours' use of the device each day, at first, the time gradually increased as better tolerance is established, is far more satisfactory.

One benefit from a good hearing device is that the patient retains a normal voice from its correct, continued use. It is a matter of common observation, that those who hear spoken words badly very soon speak those words incorrectly and strangely. Any device that enables the patient to hear the spoken word and the quality of the speaker's voice, will cause him to retain his own voice at or near its normal.

All modern experience and observation has shown the great value of lip-reading. Many cities have thoroughly organized classes for teaching lip-reading to the hard of hearing, which are doing surprisingly good work under the national guidance of experienced teachers like Dr. Newhart. All cities should have such classes, both in the public schools and privately, to care for adults past school age. It is the duty of otologists everywhere to take active interest in these associations, and to encourage their promotion in cities and towns not already provided, for it is certain that proper help for the hard of hearing will come at its best only when otologists, who know more about the defects of hearing than others, do all that is in them to help these unfortunate people, who too often struggle in vain to help themselves.

Dr. L. W. Jessaman, Framingham, Mass.: I do not think there is any particular disagreement among us as to the value of lip-reading and mechanical devices, but there are some other things that have not been touched upon that this Society, being the largest organization of specialists in the country, could do. That is, do some missionary work, especially with the idea of helping the children—do missionary work among school authorities and also among the people in general, to get them to understand the value of lip-reading, how much

it will mean to the child who is partially deaf and has to sit in the front seat in the schoolroom in order to hear. That child is tremendously handicapped, not only in school, but also outside in everyday associations. If these children have been taught lip-reading, they can pass as practically normal individuals as to their hearing. We as individuals could carry something of this sort in our minds, not only thinking of the adult, but of the smaller child. We also need teachers of lip-reading, not only enough to supply the great cities, but also the smaller ones, so when we tell parents that a child should be taught lip-reading, it can have the instruction without having to be sent too far away from home. Those of you who live in the large cities, perhaps, will not have so much of that, because now in many places they have well organized classes, taught by men from larger centers; but in the smaller places, where you cannot find a teacher of lip-reading, it is hard to get anyone to come into the locality to give instruction. In other words, it is largely a lack of organization. Let us keep that in mind as we go about in our daily practice, and not only try to handle our individual cases, but also try to get school authorities to do something to educate the public.

DR. HORACE NEWHART, Minneapolis, Minn. (closing): I wish to thank those who have so generously taken part in the discussion and helped to amplify this subject, which to me looms large.

Mention should be made of the fact that in nearly all of our larger cities we have organizations for the hard of hearing known as leagues, lip-reading clubs, or speech-reading clubs. These organizations take up the work for the hard of hearing where the physicians's work, in the past, has left off.

The handicapped hard of hearing are in a class by themselves; they are gloomy, depressed and discouraged, many of them developing early a deficiency complex. Not a few are economically in a bad way. These organizations make it their chief object to help restore these people as nearly as possible to normalcy. In other words, they look after their economic, education and social rehabilitation. Otologists do not realize how much help the organizations for the hard of hearing can render the medical man in the care of his hard of hearing patient nor do they appreciate how eagerly these groups welcome the co-operation and interest of the otologist. The activities of these local groups, co-ordinated through the national body, the American Federation of Organizations for the Hard of Hearing, constitute a very potent aid for the hard of hearing.

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We have recently been advised of the American itinerary of Dr. Franz Hasslinger, of Vienna. Dr. Hasslinger arrived in New York February 26, and gave his first course in Cleveland. His next stop was Buffalo and at the present writing he has just concluded his course in Kansas City. He is due to demonstrate his *"Phantom"* on April 1, before the Chicago Laryngological Society and starts his Chicago course the next day. After a week's course in Chicago arrangements have been made for courses in Milwaukee, Detroit and St. Paul. About May 1, he will deliver a course either in St. Louis or Dallas, then he gives a second course in Kansas City which has been arranged by the state. From there to Cincinnati, and then finally, New York will terminate his lecture course in America.

The local societies in the various cities are in charge of these courses and anyone desiring further information should communicate with the secretary of the local medical society.

Grain, in the *Presse Medicale*, Jan. 12, 1929, recommends the local and general use of vaccinotherapy in the treatment of nasal suppurations. Where possible he uses local application of autogenous vaccines; where this is unobtainable he uses polyvalent antivirus. The preparation is applied to the nasal and pharyngeal mucous membranes and is combined with general vaccinotherapy. The author claims good results in even chronic protracted sinusitis. Where he obtains no results from this therapy he deduces that the mucous membrane is completely destroyed and uses this deduction as his indication for surgical treatment. He condemns as useless and even harmful the general antiseptic applications so universally in use.

Announcement has been received of a postgraduate course given by the faculty of medicine of the University of Strasbourg, France. The course will be given by Prof. George C. Canuyt, July 1-13,

1929; it will be essentially practical and surgical. The participants will be given opportunity for individual examination and treatment. Attention will be paid to the use of local and regional anesthesia as well as the various methods and technique.

The system to be adopted will be: *1.* A written lecture; *2.* a screen projection of the procedure; *3.* the actual execution on the patient and then, finally, every participant will perform the operation on the cadaver.

For all other information apply to Prof. George C. Canuyt, care of Faculty of Medicine, University of Strasbourg, France.

It is interesting to know that in the last medical census of the city of Budapest there were 86 certified Ear, Nose and Throat Surgeons which is the equivalent of one specialist to every 11,500 inhabitants.

The Rhinolaryngologists, of Hungary, as a body, have petitioned the government to bring in some new legislation to control the sale of lye and other caustic poisons. At the present time the number of suicides due to lye and caustic poisoning is tremendously high and it is attributed to the uncontrolled sale of these substances.

The Influenza epidemic, in Hungary, reached its high point about the middle of February. Following in its wake there were numerous ear complications which in the majority of cases ran a very quiet course. Most cases healed after paracentesis. Especially in children, much attention had to be paid to the general condition of the patients as the Influenzal pneumonia had left them in a very weakened condition. Most of the complications were bilateral but very few came to the mastoid operation. Just about the time that the epidemic had run its course, numerous nasal sinus complications arose which were of the tendency to become chronic. They were of the hemorrhagic type and it is yet too soon to tell just what the full prognosis will be in these cases. This much, however, is certain, that during the Influenza epidemic numerous exacerbations of chronic sinus disease occurred; far more than had ever before been noted in an epidemic.

Ivy, of Chicago, in the February, 1929, Archives of Otolaryngology, presents a paper outlining the present status of the knowledge of vestibular nystagmus. He bases his work very largely on the research of Maxwell; he also reports the results of stimulation of the various canals separately and compares the vestibular apparatus of man to that of the dogfish. He has no knowledge to impart concerning the pathway of the impulses. In summing up, the author

states that deviation of the eyes is due entirely to stimulation of the labyrinth and that nystagmus is most probably due to some reflex traveling via the muscle centers of the eye. This last statement precludes the old idea of a cerebral reflex arc.

Means, of Boston, in the Journal A. M. A., of February 23, 1929, suggests a rather simple technique for steam inhalation. He takes a pitcher containing the boiling fluid and carefully blocks off the vapor by padding the mouth of the pitcher with towels and instructing the patient to breathe through a large glass tube which has been fixed above the fluid level in the pitcher. He claims that this method is more efficacious because of no skin irritation from the hot vapor. The method is in use at the Massachusetts General Hospital.

Karpow, of Saratow, in the Monatsschrift fur Ohrenheilkunde u. Laryngo-Rhinologie, January, 1929, reports research on the relationship of maxillary antrum disease and the morphological blood picture. The results of his investigations are as follows: In a purulent antrum one gets a leukocytosis and an increase of neutrophils. In mild inflammations (catarrhal conditions), there is a lymphocytosis and a diminished number of neutrophils. The author claims that the blood picture is of great use in diagnosis and prognosis of maxillary antrum disease.

Goldinberg, of Warsaw, in the Monatsschrift fur Ohrenheilkunde u. Laryn-Rhinologie, January, 1929, reports a rare finding in the internal ear capsule in a case of congenital deafness. While working on some of Professor Alexander's specimens in Vienna, he found the temporal bone of a 31-year-old woman who died of tuberculosis. Microscopically the internal ear showed atrophy of the acoustic nerve and in the neighborhood of the footplate of the stapes were to be seen typical otosclerotic foci. The same changes in varying degree were seen at the base of the cochlea and the internal auditory meatus. He concludes his article by asking just what the relationship may be between the tuberculosis of the patient, the congenital deafness and the otosclerotic foci.

Rose and Persky, of Philadelphia, in the Annals of Otology, Rhinology and Laryngology, December, 1928, report a case of Acetanilid addiction with chronic nasal disease as the cause. After operation the headache ceased and the patient gave up acetanilid without difficulty. While under the addiction the patient took one ounce of bromoseltzer daily which is the equivalent of about 240

grams of acetanilid weekly. The patient had become dyspneic and suffered severe gastric symptoms and all of his mucosae were of cyanotic blue. All symptoms rapidly disappeared on withdrawal of the drug.

Munro Cameron, of Glasgow, Scotland, in the British Journal of Laryngology and Otology, March, 1929, presents a very interesting article on Dysphagia and Anemia. This is a symptom complex which is not exceptionally rare and it undoubtedly has been missed by a vast number of the profession. The symptom complex occurs mostly in females between the ages of 39 and 60 and is due to a spasm or the relaxation of the crico-pharyngeous muscle. The mucous membrane of the throat becomes pale, dry and atrophic. The papillae of the tongue become atrophic and there is also an associated splenomegaly. There is a very marked anemia associated with the condition which is of relative type. The point that Munro Cameron brings out is that this symptom complex is based on a hysteroneurotic condition. He found that in routine working-up of his patient stomach lavage and passing of the esophagoscope always cured the condition. This statement was backed by Brown-Kelly, Logan Turner and J. S. Fraser in the discussion. No reason is given for the efficacy of the treatment.

Borovsky, of Chicago, in a recent paper read before the Chicago Pediatric Society, discussed the ear complications in meningococcus meningitis with an analysis of 190 cases. On re-examinatoin of 61 of the recovered patients 2 to 16 months after discharge from hospital it was found that 16, or 25.4 per cent, had developed deafness.

Twelve of these patients had a bilateral total deafness while the other four had a complete loss of hearing in only one ear. In two instances the deafness did not make its appearance until two and one-half and three months, respectively, after discharge from the hospital. Both of these were bilateral cases and the patients had normal hearing on discharge from the hospital. This could be explained only by scar-tissue contraction about the eighth nerve resulting in atrophic nerve changes.

Never was the slightest improvement in hearing noted in any of these cases.

In only four cases of this series of 190 was suppurative otitis media noted as a complication. Two of these patients recovered and two died.

Tilley described several cases of reflex otalgia at the meeting of the Otological Section, Royal Society of Medicine, March 1, 1929, and laid stress on otalgia due to nasal conditions, particularly sphenoid sinusitis. He pointed out that the pain was referred, like intestinal pain, through a definite "nerve circuit": there was the visceral nerve path leading to a ganglion, and a somatic path from this ganglion to the part where pain was felt. Mr. Tilley stated that the nerve path was probably: *1. efferent* from the sphenopalatine ganglion via the great superficial petrosal nerve to the geniculate ganglion; *2. afferent* from this ganglion via the "Ramus Cutaneous Facialis" to the auricular branch of the vagus. The "Ramus Cutaneous Facialis" had been found by Rhinehart in the mouse, and by Larsell and Fenton in the human fetus, but it had never been isolated in adult man. The speaker had no doubt that this nerve was present in cases of referred nasal otalgia. During discussion Mr. Gray stated that he had found this nerve in the sheep.

Dr. Dan McKenzie, at the March 1st meet of the Otological Section, Royal Society of Medicine, after discussing primary bulb thrombosis through the floor of the middle ear and cases secondary to lateral sinus thrombosis, raised the following points: *1.* Infected clot should not be curetted out of the bulb from above unless a projecting ridge of bone at the junction of sinus and bulb were first removed with a gouge. *2.* It was a sound procedure to leave no ligature on the upper end of the divided jugular vein, for the upper portion acted as a natural drain for the bulb. *3.* Where possible it was advisable to ligature and divide the jugular vein above the entrance of the common facial vein and to leave the latter untouched so that stasis of venous blood in anastomotic channels was less likely (facial veins—cavernous sinus—petrosal sinus). The reason for this was that most of the speaker's fatal cases had had cavernous sinus thrombosis.

During discussion the trend of opinion was: *1.* That radical operations on the jugular bulb were unnecessary and that leaving open the upper part of the divided jugular was a sound procedure. *2.* It was unnecessary to remove the spur of bone referred to by Dr. McKenzie and there was a danger of it falling into the bulb where it could not be easily recovered. *3.* Trouble usually followed if Dr. McKenzie's site of ligature was adopted. The most favored procedure was to ligature the jugular below the entrance of the common facial vein and in addition to tie this latter vein.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLOGY.

November 9, 1928.

Syndrome Complex, Meniere. Dr. Dana W. Drury.

(Published in March, 1929, issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. RANDAL HOYT: I think Dr. Drury's paper is very important because it emphasizes the necessity of extending our field of investigation of the various vertiginous reactions beyond the narrow confines of the labyrinth and its receptor neurons.

The labyrinth is a nerve end-organ, and it is a mistake ever to forget that a nerve end-organ is merely one of the details of an elaborate neuro-functional mechanism. Whenever any part of this mechanism is injured, the symptoms produced resemble those which occur when the end-organ itself is diseased; and, conversely, when a patient complains of such symptoms the *entire* system must be investigated before a diagnosis can be made. I think we should go even further and search the entire body for causes of those pathological processes, the location of which in the nervous system is responsible for the symptomatology.

Stimulation of the semicircular canals produces such dramatic and consistent effects that the association between vertigo and the labyrinthine dysfunction has become so fixed that when we hear the complaint of vertigo we are prone to jump to the a priori conclusion that the labyrinth or vestibular nerve must be diseased. In fact, we have become so prejudiced in this regard that even though pathological evidence shows that the labyrinth is not diseased, nevertheless we proceed as though this organ and this alone were responsible for the Meniere syndrome. There is nothing more conclusive than the evidence of Politzer which Dr. Drury quotes, for it cites cases in which patients having Meniere's syndrome did not show any post-mortem labyrinthine pathology, and other cases in which the labyrinthine pathology was found at autopsy, though during life these patients did not show the Meniere symptom-complex. Why, therefore, should we not trust our senses and reason and give up the conception of the labyrinthine etiology of this particular syndrome and look elsewhere in the vestibular mechanism for the site of the process?

If we trace the auditory nerve inward to the brain, we find that it gives the appearance of being split into its two cardinal divisions by the restiform body or inferior cerebellar peduncle. The cochlear division curves around the lateral and dorsal surfaces of this body; the vestibular division pierces the brain stem between the restiform body externally and the descending nucleus of the trigeminal nerve inward. It then continues its dorso-mesial course until it reaches the floor of the fourth ventricle. There the great bulk of the vestibular nerve divides into ascending and descending branches which terminate, respectively, in the ascending and descending vestibular nuclei. I have said that the great majority of the vestibular fibres pursue this course, in order to destroy the conception that many have that the connection between the vestibular nerve and the cerebellum is anatomically important. It is true that some vestibular fibres reach the cerebellum by the Milpole peduncle; it is equally true that there is a connection between the room nuclei of the cerebellum and Deiter's nucleus; but when one looks for these fibres microscopically they are few and far between and, in fact, so sparse that no less an authority than Ramon Y Cajal pauses to comment about it.

The vestibular nuclei occupy the greater part of the area below the floor of the fourth ventricle; they are separated from the midline by the posterior longitudinal fasiculus, which consists chiefly of secondary vestibular neurons

which pass from the vestibular nuclei to the motor nuclei of the ocular muscles. Ventral to these nuclei and at some distance from them are found the superior olives, the trapezoid nucleus, and the neurons which form the lateral fillet. These structures are all concerned with the cephaloid projection of the auditory impulses. Thus, the greater part of the brain stem which lies between the pons varolii ventrally and the fourth ventricle dorsally is largely given over to structures related to vestibular and auditory functions.

There is another peculiarity about this particular region, in that it is studded with numerous vascular lacunae. I have never been able to determine with any satisfaction what these are, whether they are arteries or sinuses. They are, however, numerous and relatively large, and I have no doubt but that if this particular part of the nervous system could be inspected during life the same fluctuations between pallor and hyperemia could be observed in it as are sometimes observed in the nasal mucous membrane. It is very significant to me because, in the first place, it seems to show how the vascular balance in this region could be affected by endocrine disturbances, or how hemic toxins could readily affect the surrounding parenchyma. In other words, when we correlate the facts which I have tried to describe, we find that on account of its peculiar vascularity, and on account of the fact that this particular part of the brain is given over to the auditory and vestibular functions, it is the locus ideal for those pathological processes which cause the Meniere symptom-complex. A lesion in this particular region could easily explain why the symptoms of this complex are bilateral; it explains why though bilateral they are not necessarily symmetrical—that is, why tinnitus or deafness is greater on one side than the other; and, again, in a large area like this, one can understand why when the pathological process disappears it leaves auditory or vestibular symptoms as residuals. Finally, it explains how by extension the disease can involve the nuclei of the ocular muscles and produce the diplopia which Dr. Drury mentioned.

I feel that the broad biological point of view from which Dr. Drury regards the Meniere symptom-complex is a definite step in advance, and I feel that it will stimulate investigation of the vestibular mechanism as a whole, the result of which I hope will dispel the uncertainty which now prevails concerning the cause of this syndrome.

J. D. WHITHAM: The endocrine and theoretical aspects of this subject are so far above the heads of most of us that I hesitate to discuss it. Dr. Drury's paper is, of course, chiefly on the Meniere syndrome, but he showed records of some 500 cases. Now, it seems unlikely that he or anyone else has seen so many cases showing a close resemblance to that syndrome.

There are so many causes of the labyrinthine type of vertigo that have not been mentioned that I though it might be of interest to mention a few of them:

1. Vertigo in affections of the external auditory canal. Example, impacted cerumen.

2. Vertigo in inflammatory affections of the ear, as hydrops of the labyrinth, serous or purulent labyrinthitis, fistula necrosis or empyema of the endolymphatic sac.

3. Vertigo in O.M.P.C. residual.

4. Vertigo in O.M.P.C. and tubal conditions.

5. Vertigo in otosclerosis.

6. Vertigo in syphilis of the labyrinth. Diffuse and of sudden onset in the secondary stage and usually of slow progress in the tertiary stage. I have seen two cases of secondary syphilis with the Meniere syndrome. The vertigo and tinnitus were most severe and deafness was profound, but recovery was almost complete after treatment. Tuberculosis, typhoid fever and malaria have been named as causes.

7. Vertigo in intoxications of the labyrinth. (a) Exogenous, as tobacco, alcohol, salicylates, quinin, drugs, etc. I noted that some of Dr. Drury's cases had had quinin and that may have caused some damage to the vestibular cochlear apparatus. (b) Endogenous, as intestinal, hepatic, gouty and focal infections.

8. Vertigo in circulatory troubles of the ear, hyperemia of anemia of the labyrinth. Hautant thinks that the vertigo of Meniere is due to an angio-

neurotic crisis in an ear already altered by a sclerosing process. In this connection anemia, vascular and cardiac conditions, especially leukemia, should be mentioned. The first case of Meniere is now thought to have been a leukemic intra-labyrinthine hemorrhage.

9. Vertigo in disturbances of tension in the internal ear. Sudden increase or decrease in the intracranial pressure in the posterior fossa by causing a rise or fall of pressure in the endolymphatic sac or the vestibular aqueduct may determine a typical Meniere syndrome. Such a state of affairs is vaso-secretory or sympathetic in origin.

10. Vertigo after cranial trauma. This type of vertigo is usually associated with deafness and spontaneous nystagmus.

11. Vertigo of sympathetic origin. As an irritative lesion in the realm of the pneumogastric or trifacial nerve, or in patients with endocrine disorders, as so ably brought out by Dr. Drury.

This brief outline is freely quoted from the recent valuable work of Moulonguet on labyrinthine vertigo. As for treatment, I do not believe that endocrine disorders could possibly account for more than a very small fraction of cases of vertigo. The use of laxatives, intestinal antiseptics, mercury, atropine, pilocarpine or nitrites in suitable selected cases associated with the proper local ear treatment must be given its place.

The surgical treatment is reserved for the severest cases. The following operations have been devised: trepanation of the labyrinth antefacial or postfacial, incision of the cerebellar dura, incision of the endolymphatic sac and section of the auditory nerve. All of these procedures are very difficult, dangerous to life and uncertain in their results.

DR. CLARENCE H. SMITH: As I understood Dr. Drury, he advances the idea that the secretions of the endocrine glands at times are toxic and I can very well understand how they would be so; but I rather think they would produce their results slowly and in a chronic way and this would not altogether explain those cases presenting the symptoms of upset of equilibration sharply and in the fulminating way we see every now and then. I remember the case of a young woman who was suddenly taken sick while eating a Christmas dinner, with an attack of intense vertigo and disturbance of equilibrium, and was put to bed. I saw her within a few days and her hearing was gone in one ear. At that time she had vertigo and trouble with her equilibrium. She had no high blood pressure, and there was nothing apparent to show the cause of it; I did not go into the question of endocrines, and my explanation was that the symptoms were probably due to a hemorrhage into the labyrinth. Why she should have had it I did not know, but that was the best way I could account for it. One bad thing I have seen is that the deafness is permanent.

Dr. Hoyt's explanation is very interesting and his illuminating talk about the anatomy is helpful. I would like to ask Dr. Drury if in closing he would give his views on the very sudden onset in these cases.

DR. JOHN GUTTMAN: I do not believe that there is a great divergence of opinion between the essayist and the gentlemen who discussed the paper. While the essayist considered in his paper mostly the affections of the peripheral vestibular apparatus, Dr. Hoyt emphasized mostly the central vestibular system and gave a masterly description of the anatomy of this part of the brain.

Dr. Drury described the classical symptoms of a chronic non-suppurative inner ear affection, consisting of vertigo and deafness; he failed to mention the objective symptom of vertigo—disturbance of equilibrium—which must have been present in some of his cases. Deafness is also a rather vague term, as this may include a condition ranging from diminished hearing to stone deafness. In several of his cases he mentions the fact that the patient was pale and that the symptoms became aggravated by sudden motions, as bending, etc. These symptoms are quite characteristic for labyrinthine affections. He considered as the main etiologic factor in the majority of his cases of inner ear disease, some endocrine disturbance. He mentioned cases which he cured with thyroid extract. I do not believe that many of us would subscribe to this, since many other factors, such as circulatory disturbances or intoxications, could be held responsible for these affections. I am treating at present a similar case to one

described by the essayist, a doctor who lived in the Orient and took a great deal of quinin for malaria, is at present also suffering from nausea, vertigo, and diminished hearing; an intoxication of his vestibular system by quinin explains fully the etiology of his affection.

All in all, the paper, as well as the discussion, was very interesting.

DR. DRURY: I wish to thank the men for the interesting discussion. In the way of an explanation I would say that I judged a paper of 40 minutes would about fill the bill. Having never seen in English a complete translation of Meniere's cases and only the isolated case that died and was autopsied, I thought such an arrangement would be more to your liking than a set of audiograms and the minutiae of the several cases.

All cases are not of thyroid origin in this syndrome. The pituitary is involved also. Again we meet cases of non-endocrine origin. Betzold, of Munich, before he died, told me that he considered the patient of Meniere a case of cerebro-spinal meningitis. We should not make a diagnosis solely upon a basal rate showing a minus percentage. Again, it is always wise to have more than one basal measurement.

The two tables of course were not all Meniere's disease. They were a group study showing as presenting symptoms deafness, tinnitus and vertigo. I do not think any single test can be regarded as significant unless checked and controlled by a number of independent observations. Again, no single finding suggestive of an endocrine disease should be made the basis of such a diagnosis until other causes have been ruled out by observation and tests.

SECTION OF LARYNGOLOGY AND RHINOLOGY.

November 27, 1928.

Lung Abscess. Some Aspects of Etiology and Medical Treatment. Dr. James A. Miller.

(Published in full in this issue.)

DISCUSSION.

DR. T. J. HARRIS: It is indeed an honor and a privilege to speak a word for the Advisory Committee of the Section of Laryngology and Rhinology. As I think most of you are aware, last spring, as the result of very long deliberations by a special committee appointed by the Council of the Academy, the plan of Advisory Committees to the Sections was adopted, among other recommendations; and the Advisory Committee of this Section has had the privilege of serving with your Chairman.

I want to say for the Chairman that we have nothing but words of admiration for the way he is accomplishing things. The time and thought that he has put in for the work of the Section is truly phenomenal. I have had the opportunity of observing many chairmen, and I know of no one who has spent more time and thought upon the work that the present Chairman.

The idea of a dinner is a most happy one. The bringing out the attendance, as Dr. Mayer brought out the opportunities for social intercourse,—all make for the best interests of the Section. For many years this Section had the largest attendance of any Section of the Academy, and it has been the mouthpiece of many men whe have risen from the ranks to the top of their profession. It affords the younger men an opportunity to be heard. I was particularly interested in hearing Dr. Emil Mayer's letter tonight, and I would suggest that it would be very fitting and suitable if our Secretary would reply to Dr. Mayer for the Section and express to him our appreciation and best wishes and hope for a complete and early recovery. No one has done more for Laryngology than Dr. Mayer, and no one would more appreciate such a greeting.

I congratulate you, Mr. Chairman, on what you have done so far, and I wish for you the same degree of success that you have scored tonight.

DR. C. J. IMPERATORI: I doubt very much if I can add anything to the scientific value of this discussion. The mechanism of the production of lung abscess is not clear. Dr. Fetterolf's experiments on the production of lung abscess are very interesting, but not conclusive. I do not think we have as yet scientifically determined the method of the invasion of the lung and the formation of a lung abscess. Bacteriologically, it would seem that anaerobic invasion of the lung substance is the cause of lung abscess; this has been my opinion for many years. When working in Bellevue Hospital about the time Dr. Miller and Dr. Lambert became interested in this subject, I unfortunately bronchoscoped a man who died. Following the bronchoscopy, he walked down five flights of stairs, and later died from a hemorrhage. At that time we had quite a series of cases, and I was of the opinion that the chronic lung abscess was different from what we had previously thought, and autopsies showed that my conception was correct.

I will read a few notes from a paper prepared in 1921 on this subject, embodying observations from the service of Dr. Coakley of seven cases. Two lived following treatment; five died; one was a case of carcinoma of the bronchus; another died following a pneumomectomy, dieing on the table; and the others died from inter-current pneumonia. The autopsy findings were interesting. They showed a periphery of tubercles surrounding the abscesses. Examinations of the sputum had been done without coming to any conclusion; that is, no tubercle bacilli were found after repeated (25 to 30 times) examinations. At autopsy, the five cases showed tubercles surrounding the abscesses.

Insofar as the diagnosis goes, I feel that the problem is as has been outlined by all the speakers.

In regard to treatment, it is entirely empirical, and follows our past experiences of the combined methods of posture, bronchoscopic and surgical treatments.

I have never felt that I could enter a lung abscess cavity with the bronchoscope. Diagnostic bronchoscopy, aspiration and after reaching the bronchus from which most of the pus appears to come, instillation of arsenical solution—seems to give decided results, in acute cases, improvement being noted at once. The patient is instructed in regard to the postural position, and where the hospital is equipped with mechanical beds, the patient can take the postural position with the least discomfort. It is not easy for the patient to take these postural positions, even with the best apparatus. In my experience, the ordinary bed tipped up has been very successful. The patient is instructed in the use of oxygen inhalations. With this method, postural drainage, occasional bronchoscopy, and the use of oxygen, results have been fair.

I was delighted to hear Dr. Lambert make the statement that a lung abscess is very difficult to find surgically, but I hope at some future time to report something more scientific regarding the localization of the lung abscess. Ten years ago, Dr. Lynah, who did a great deal of this work and was among the first to outline a lung abscess by injecting bismuth oil, in a discussion on this subject made the statement that in his opinion the use of bismuth, which he was employing at that time, had a dilating effect on strictures. I think he had absolutely the right idea. If we could ventilate these cavities at the same time that we drain them, I think we would go a long way toward curing them.

DR. J. D. KERNAN: I realize very fully that I would be an unusually smart man to say anything new after all the discussion we have had tonight. I suppose this meeting is the culmination of something that most of us who have been interested in the subject of lung abscess for a number of years have been working for—that is, the co-operation of the medical man, the bronchoscopist, the surgeon and the roentgenologist in the diagnosis and treatment of lung abscess. My remarks will be very disconnected, I am sorry to say, but I would like to go over the high points in the remarks of each of these gentlemen.

I was interested in what Dr. Miller said of the importance of sinuses in connection with the etiology of lung abscess; especially are the sinuses important in connection with bronchiectasis—and his remarks on the bacteriology of the mouth.

As to the two schools which have been in the past, and still are, fighting over the question as to whether lung abscesses are caused by aspiration or by emboli, I can cite one case of a lung abscess following a tonsillectomy, in which I removed a piece of tonsil from the lung abscess about two weeks after the tonsillectomy. The tissue diagnosis was made under the microscope —so that in one case at least we have positive evidence of a lung abscess caused by aspiration. I have no doubt that many lung abscesses are also caused by emboli.

I was surprised to hear Dr. Miller say that 50 per cent reached the surgeon; I should have supposed that not more than 25 per cent did so. My idea of the treatment of lung abscess is that every case deserves at least one bronchoscopy; first, on the chance of there being a foreign body in the lung; and second, on the chance of the case getting well with one bronchoscopy. It cannot be prophesied beforehand just what effect the bronchoscopy will have on the disease. I have seen a case of lung abscess get well in which there appeared to be an inter-lobar empyema; and I have seen a lung abscess close to the hilus, which was thought to be easy to cure, fail to get well. Many of the tonsillectomy cases get well remarkably after bronchoscopy, in the manner in which foreign body cases get well, with a crisis. That is one reason why I think many of the tonsil cases are due to aspiration.

I wish Dr. Miller would say a word about lipiodol. It would seem that at times lipiodol has rather increased the size of the abscess and made the picture worse.

As to postural drainage, I would like to ask Dr. Miller if he thinks there is any danger of involving the other lung. I have seen a case in which, following an abscess in the left lower lobe treated by bronchoscopy and postural drainage, and finally handed over to the surgeon and ending in autopsy, a large abscess was disclosed in the right lung, which had not been suspected.

I think that my experience has been a little different from Dr. Yankauer's. It has always seemed to me the position of the abscess, near the hilus or far away, the size of the abscess, and whether multiple or not, has much to do with the success of bronchoscopy; also, I cannot understand how a bronchus can enter the outside of an abscess cavity, radiating from the hilus toward the periphery.

DR. YANKAUER: As to the length of time to persist in bronchoscopy: I know that cases do get well with bronchoscopy when persisted in for a long time. Dr. Lynah had a case bronchoscoped for two years, which ended in complete cure, both symptomatically and according to X-ray findings. As a rule, such persistence will do harm. I have recently had a case which was bronchoscoped, and at first, improved. Then the patient had a recurrence which seemed to have been caused by the formation of scar tissue. The very healing process seemed to make him worse. The bronchus was blocked proximal the abscess cavity, and the retention of pus necessitated a surgical operation, made all the more difficult by being delayed so long.

Dr. LeWald's pictures speak for themselves.

Never before did I realize that patients could survive after the complete destruction of an entire lung by suppuration. The lesson to be learned from these cases is the necessity for early diagnosis and cure of lung abscess, for if allowed to go on, even though the patient survives, he becomes a wretched invalid.

Replying to Dr. Lambert, the question of surgical judgment here arises. When should the patient be operated? We know that too early an operation is very dangerous, and yet Dr. Lambert is right in saying that it takes immense courage at times to refuse an operation.

As shown by the case just quoted, too long a delay in operating makes the operation difficult, and sometimes makes a cure, even by surgical means, impossible.

Dr. Miller and Dr. Lambert give a definite rule as to time of operation; that is to say, about two months from the time the abscess began.

The matter is not quite so simple as that. Some patients require early operation and some will get well without operation, even if it is delayed much longer than two months. The answer to the question is in having a competent

surgical consultant from the first. Need I tell you that we have one with us tonight?

DR. MILLER: I have nothing to add, but will answer some of the questions. In the first place, I would like to emphasize that what we are trying to do is to get at the abscesses before they become chronic. I think the treatment of chronic abscesses is extremely unsatisfactory; many of the cases which Dr. LeWald showed must have been of long standing and very difficult to cure by any method.

As to tuberculosis: I have never been impressed with the association between lung abscesses and tuberculosis. There is no doubt that the chronic form in association with tuberculosis may and does occur. Upon one or two occasions I have recommended operation on cases for lung abscess which turned out to be tuberculosis. It was purely a fault in the diagnosis.

As to the danger of posture and the danger of secondary abscess: Of course secondary abscesses do occur, but I never thought the posture was responsible for them. On the other hand, I am sure that lipiodol injections do tend to cause these secondary abscesses. Consequently, as Dr. LeWald showed, there is very little useful information to be obtained by this method and I think that it is unwise to us it at the present time in pulmonary abscess.

One other point I want to emphasize—that after you have patients free from symptoms they are apt to relapse unless all vestige of infection has been cleared up, and that means time. I have had a number of patients go back to work against my advice, who still had a very slight amount of lesion in the lung; such cases again develop abscesses due to premature return to active life. That is like treating a case of tuberculosis—a long rest cure before letting them go back to work in order to obtain permanent results.

I plead that we ought not to think of treating this disease by any one method. Up to the last few years we have of the results obtained by medical treatment, artificial pneumothorax, by bronchoscopy or by surgery, but if you make up your mind that any one of these methods is the only right method you will be wrong, for all will fail at times, though all of them may be useful for certain types of the disease. Co-operative study and treatment is the modern method, and this symposium, bringing together the various interests involved, is hopeful evidence that it is being appreciated.

DR. YANKAUER: I wish to add my own word to what I think has been the principal result of the present discussion, bringing out the necessity of co-operation between the different men who are interested in this subject. We have recently formed such a co-operative unit at Mt. Sinai Hospital among the surgeons, the internes, the bronchoscopists and the roentgenologists for studying every case. Such a group has been established for some time in Boston and has been very successful. I think this is a very definite demonstration of the value of this combined method of study.

DR. LAMBERT: It is always a joy to have a paper discussed by Dr. Kernan. When he got up he said he would be a smart man if he could add anything to the discussion, and I think his remarks rather showed he was a smart man.

Lung Abscess; Roentgenologic Diagnosis. Dr. Leon Theodore LeWald.

This presentation was in the form of a series of roentgen pictures to illustrate the points in question. Unfortunately it is impossible to reproduce Dr. LeWald's contribution to the symposium.

Bronchoscopic Diagnosis and Treatment of Lung Abscess. Dr. Sidney Yankauer.

(*Published in full in this issue.*)

Surgical Diagnosis and Treatment of Lung Abscess. Dr. Adrian V. S. Lambert.

(*Published in full in this issue.*)

SECTION OF OTOLOGY.

December 14, 1928.

Contribution to the Surgical Indications in Purulent Labyrinthitis and Otitic Meningitis. Dr. Samuel J. Kopetzky and Dr. Ralph Almour.

(*Published in full in this issue.*)

DISCUSSION.

DR. WELLS P. EAGLETON: Both Dr. Kopetzky and Dr. Almour kindly sent me their paper, covering in many ways the whole subject of labyrinthitis in relation to meningitis, and I read them with a great deal of interest. I am grateful for the compliment Dr. Kopetzky pays my work.

Dr. Kopetzky spoke of trauma.

The study of fracture of the skull and meningitis following fracture of the skull have been the greatest factor in my understanding of meningitis. When Dr. Brunner was in this country he called my attention to the mechanism of the fracture, and since then we have learned to follow the line of fracture, no matter where it goes, as soon as we have meningeal symptoms, and drain; our results have been good.

If we take that principle and apply it to all our otological cases we can get many of them well. If we will diagnose the way the infection enters the meninges (for in the majority of cases of meningitis the infection is limited for a long time to the area of the pia-arachnoid that has first been attacked), by the infective process, and it is simply a matter of diagnosis on an anatomical basis.

Meningitis, in my experience, follows definite tracks, depending on the lesion itself.

First of all, it enters by the veins. There has never been any complete study of the veins of the petrous pyramid; at least I have never been able to find one—and yet my anatomical dissections would show that there are very definite small veins. There are two distinct plexuses that enter the triangle; they go into the big sinuses. One goes into the tegmen; one into the petrous pyramid.

If you take an anatomical specimen that has been injected, the veins can be studied as they pass through the dura into the pia and arachnoid. Many of the cases follow these small veins, and as our study of diagnosis and our knowledge of anatomy become improved, we shall be able to recognize the venous infections which today we are disregarding.

I have with me the last two meninges of patients who have died and came to post-mortem. Two of these cases showed the influence of infection into the subdural space.

Second, infection also goes from the petrous pyramid directly into the pia-arachnoid, the arachnoid prolongations and into the temporal bones are very definite; the ductus lymphaticus, the triangle, the internal auditory meatus, the tegmen. Then we have dehiscences, either congenital or acquired. If you study the patient's history and the otological findings, and find these tracks, you will be able to go into definite areas of the pia arachnoid. After entering the arachnoid, nature walls off the infected area at least for a time, and if we find and evacuate the fluid of that area, not all the fluid of the whole cerebro-spinal system, but that which is infected, results will be much better.

Dr. Almour's statement that purulent and serous labyrinthitis will have a high temperature is one that I cannot agree with. In a serous labyrinthitis there is no temperature; they may have a high temperature from the disease that caused the serous labyrinthitis, but none from the labyrinthitis itself. In fact, in all our diagnoses we should take the picture as a whole, and not give any special attention to the presence or absence of any one symptom.

Again, Dr. Kopetzky speaks of the Gradenigo syndrome as being seldom of importance, and is always due to a slight form of meningitis which generally recovers. This, I think is a mistaken attitude. Some years ago I spent a month studying anatomy with the idea of finding out why certain cases make

so little progress and have so little pain, while other cases act otherwise. It is very simple if we approach the subject anatomically. Because of the anatomical complications, fifth and sixth nerve pain can occur in the slightest inflammation in the dura. The inflammation may be transmitted a long distance. We should be able, by a study of fifth nerve pain and sixth nerve paralysis, to determine whether we have disease of the apex of the petrous portion, and if we have that, then we have meningitis of the basal cisterna itself and these cases always die unless operated upon and whether or not there is an associated sepsis.

One other thing: that is, the use of the chisel. I use the electric drill and burr. The reason for so many cases being lost in Vienna is because they use the hammer and chisel. I do not think you can save many cases if you take a chisel and hammer and pound on the tissue in a patient who is already tremendously prostrated by disease.

If we once realize the amount of shock in chiselling away bone over a large area on a patient who has no ether, and realize the ease and rapidity with which one can remove the whole eburnized pyramid, I am sure that no one here would ever do a mastoid operation without it, especially when the patient is debilitated by such a debilitating disease as meningitis.

DR. E. D. FRIEDMAN: I rise with some hesitation to discuss a subject in which the otologists have had a much larger experience than the neurologists. I am very grateful for the privilege of listening to these papers. To me, as a neurologist, this field has always been a difficult one. It has interested me for many years, and I recall with pleasure a few hours which I spent with Freml in Vienna in an attempt to obtain a little more light on the subject. I can say little with regard to Dr. Almour's paper. Most of us are in accord with him. There is also not much to add to what Dr. Kopetzky has said.

We must all agree with the thesis that the fundamental consideration in the treatment of otogenic meningitis is the operative removal of the primary focus in the middle ear. Although there are cases on record in which an undoubted, diffuse, purulent meningitis of otitic origin healed without operation, and although I have seen some of them heal after repeated lumbar puncture, these are the rare exceptions. It is also safe to say that in most cases of healed otogenic meningitis in which operation was carried out, the surgical interference may be held responsible for the favorable outcome. Many years ago Janset up the dictum that the first meningitis symptoms constitute the last warning to the surgeon that complete and thorough removal of the suppurating focus in the petrous bone is indicated. The operative removal of the infectious material should be as complete as possible. At the same time, other complications, such as extra and subdural abscesses and sinus thrombosis, can be dealt with. It is my belief that in every case the dura of the middle and posterior fossa should be exposed. In this way an adequate outlet for the infectious material is provided, and its conduction toward the interior of the cranium is lessened.

The conditions in otogenic meningitis are somewhat similar to those in subphrenic abscess with so-called reactive pleurisy above. In such instances the surgical attack of the focus of sub-diaphragmatic suppuration is clearly indicated, and, by analogy, it is equally in order in cases of otogenic meningitis.

The question of incision of the dura is still a moot one. If it is found seriously altered (gangrenous, perforated, tense and without pulsation), and we suspect a circumscribed pus focus behind it, it should be incised. The suggestion made by Dr. Kopetzky to determine the path of the infection and to drain accordingly is intriguing. The routes of infection are usually by way of the tegmen tympani and by way of the acoustic and facial nerves to the meninges. Exposure of the internal auditory meatus and of the dura of the middle fossa should therefore be carried out. The reasons have been ably advanced by Dr. Kopetzky.

Permanent drainage thus far has proved uncertain in its results. While it may possess the advantage of relieving intracranial pressure, it contains an element of danger, owing to the tendency to prolapse of the brain. Drainage of one of the great cisterns, however, provides better opportunities for the escape of infectious material and at the same time avoids the prolapse of the

brain. Thus far neither dural nor subarachnoid incisions have yielded sufficient drainage for the diseased subarachnoid space and meninges. Dr. Kopetzky's experiences, if corroborated, may lead to a revision of our accepted opinions on this point.

I was very much interested to hear of Dr. Eagleton's work on the differences between cultures obtained from the lumbar sac and those obtained from the site of infection. Some of you may recall that Weigelt, Ayer and others have found differences in the chemical composition of the cerebro-spinal fluid at various levels of the subarachnoid space. It may be that the circulation of this fluid is not so active as we have been led to believe. It may therefore be that the organisms at the site of infection do not freely enter the subarachnoid space. It is consequently unwise to wait for the appearance of organisms in the lumbar sac before operating in cases of otogenic meningitis. It is very difficult, in a given case, to tell whether one is dealing with a sterile meningitis sympatica or with a suppurative meningitis which is bacterial to start with. I believe, therefore, that nothing can be said against the thesis that operation should be carried out quickly and completely.

I was interested, too, to hear Dr. Kopetzky's remarks regarding the relative benignity of some types of meningitis. What he said is, in the main, true. I think, however, that I shall have to agree with Dr. Eagleton that some of the cases presenting the Gradenigo syndrome are not so benign. This is particularly the case when it occurs in children in whom the mastoid and petrous bone are very pneumatic. In such instances the advent of the Gradenigo syndrome constitutes a clear indication for surgical interference.

In conclusion, I shall cite two personal experiences:

The first case was that of a boy who was admitted to the Otological Service of Mt. Sinai Hospital in February, 1928. There was a history of chronic purulent otitis media. The patient presented the evidences of a right mastoid infection and an active meningitis. The spinal fluid was turbid, the cell count was 2,500, 90 per cent of which were polynuclears. No organisms were found on smear or culture. The blood culture was negative. The white cell count was 12,000, with polynucleosis of 63 per cent. X-ray examination revealed destructive changes in the right mastoid. Mastoidectomy was carried out. At operation there were found a perisinus abscess, an epidural abscess, and the signs of an acute mastoiditis. The patient was discharged well.

The second case was that of a young boy, who was admitted to the Neurological Service of Mt. Sinai Hospital in September, 1928. There was a history of acute otitis six weeks prior to admission. On admission it appeared almost resolved. The clinical syndrome was that of a fulminating meningitis. There were no objective signs of mastoiditis. Lumbar puncture revealed the presence of turbid fluid, with 3,200 cells, 95 per cent of which were polynuclears. There were no organisms on smear or culture. The white cell count was 44,000, 96 per cent of which were polynuclears. X-ray examination revealed a necrotic mastoid. Mastoidectomy was carried out at once. A perisinus abscess was found. The dura was exposed in the middle fossa, but found to be normal. The symptoms promptly cleared up and the patient was discharged well. Blood and spinal fluid cultures proved negative.

DR. M. S. REUBEN: In the last few years I had the opportunity to observe many of these cases. Dr. Kopetzky mentioned several things about meningitis sympatica. I think the term is a very unfortunate one, and would not have been adopted if it were not of German origin. The French term, neighborhood meningitis, is much better. If we compare the conditions which are present in the mastoid and in regions around it to a carbuncle, we can better understand the treatment in these cases. When we open a carbuncle, we do not incise the protective area around it, and yet it appears that this procedure has been suggested, and in that way destroy the best protection which nature has set up against the invasion of the meninges. In our experience a great many cases of sinus thrombosis and mastoiditis have shown signs of meningitis sympatica. In fact, I have not seen a case of sinus thrombosis which did not show it and, moreover, many cases of mastoiditis present evidence of it. It is simply nature's way of protecting the meninges. In the great majority of these cases when the primary focus is removed the symptoms of meningitis sympatica subside.

In the last two years I have had occasion to take care of six doctors' children. All of these had mastoiditis and sinus thrombosis in association with meningitis sympatica. In all of these cases the meningitis subsided after the primary operations. Three of these cases were operated by Dr. Koeptzky, and all recovered, in spite of the fact that the spinal fluid showed evidences of severe meningeal irritation. I cannot quote a series of six cases in succession which were subjected to secondary operations on the meninges without fatalities. One has to be an optimist to believe that if these cases had not been operated on they would not have recovered. The proof of the pudding in these cases would be to obtain surgical recovery in cases of meningitis following otitic conditions, in which organisms are found in the cerebro-spinal fluid. The evidence that many cases can be saved by opening the dura before organisms are found in spinal fluid is not convincing, in as much as the great majority recover without operation. One case seen recently had marked herniation of the brain, and the patient died on the table; this case was not operated by Dr. Kopetzky.

Dr. Kopetzky and Dr. Eagleton are very skillful technicians and they can get away with such treatment, but in the hands of the general otologist these cases should be let alone. Personally, I do not feel that in these cases of localized protective meningitis surgery is indicated. However, I do believe that in certain cases of generalized meningitis surgery is indicated. We recently had a case which did not respond to treatment. Dr. Kopetzky instituted permanent drainage of the cisterna magna, and in a few days there was remarkable improvement, which went on to complete recovery. In this case organisms were found in the spinal fluid. There are cases of cerebro-spinal meningitis which are amenable to surgical treatment, cases in whom we can obtain fluid on spinal puncture and in whom there are present a rising pulse and respiratory rate and rising temperature, with delirium. If these cases do not respond to simple puncture of the cisterna, they should have an open operation on the cisterna. However, the efficiency of surgery in cases of meningitis following otitic conditions must rest on the ability of this procedure to procure recovery in those cases of meningitis, in which organisms are found in the spinal fluid.

DR. PHILIP D. KERRISON: We all know that meningitis generally is one of the most hopeless diseases; even though in cases that go on to what we call a purulent meningitis, where the pus is shown in the spinal fluid certain ones do get better, most of them die. Certain ones do get better, but the majority do not.

I think the speaker who preceded me missed somewhat Dr. Kopetzky's point. I liked Dr. Kopetzky's paper because it is logical and has some very practical points. I cannot follow him in his inability to determine the course of infection at the time of operation, but in one case he certainly has a logical deduction—where you have a case of labyrinthitis and meningeal symptoms.

Labyrinthitis is one of the most puzzling conditions the otologist has to deal with; for instance, a chronic suppurative labyrinthitis, whether that should be operated on or whether a radical mastoid should be performed; but in a case of acute labyrinthitis it is most difficult for the otologist to determine what to do, because if he operates in a simple labyrinthitis there is a very great possibility of starting up a process in the meninges, with a fatal result; if he waits until the meninges are affected he may lose the patient and wish he had operated.

I think the point Dr. Kopetzy brought out, that an acute labyrinthitis should be watched very closely, and the first clinical signs of mental irritation as caused by increased pressure, is a very good one, and one that so far as I know has not been mentioned before. It gives you something to go on. As for the cases that have arrived at that stage, the preceding speaker seemed to think they might have recovered without operation. Many cases are recorded in the literature where a simple exploration has brought about recovery; but the point is, that in such cases as the first and third, if the incision was not made, the possibility, or even the probability, is that those two cases would have been converted into purulent meningitis, and it is a problem whether anything could have been done for them.

I was unable to follow Dr. Kopetzky's indications in the second case, where he operated on the cisterna magna. Dr. Eagleton makes the cheerful response that even in the operative procedure is advisable; but my personal experience is that nothing has been proved to be effective, although some cases have improved.

The point is, that the indications for operation in meningitis are the acute signs, and not to give it time to spread to a leptomeningitis.

DR. ALMOUR: I wish to thank Dr. Eagleton for his kindness to me. Dr. Reuben stated that many of these cases of meningitis where an organism is not found in the spinal fluid get well. In these cases there is meningitis sympatica, but the point that we are trying to make is that you cannot tell when a meningitis sympatica will stay one and when it will subsequently be converted into a purulent meningitis. You can always trace the stages of meningitis sympatica in brain abscess; but we feel that a purulent meningitis, when no organism is found by lumbar tap might just as possibly be the early stage of a purulent meningitis wherein organisms will subsequently appear. In our diagnostic procedure of lumbar tap we are getting fluid from one part, the entire cerebro-spinal fluid, and basing our diagnosis on fluid taken from one particular part of the system; yet organisms might be present in some other portion of the subarachnoid space, where they remain localized for a time.

QUERY: I would like to ask the rationale of the magnesium sulphate enemas.

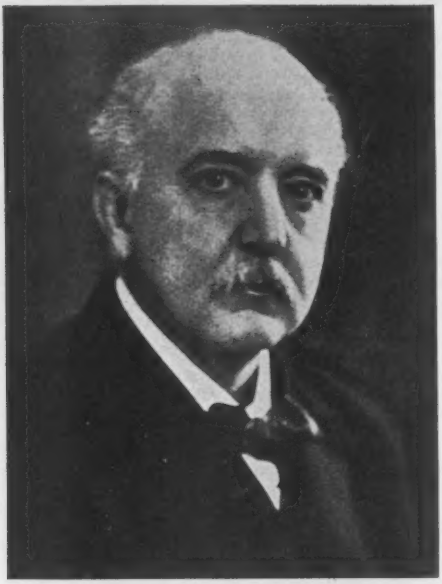
DR. KOPETZKY: I, too, wish to express my thanks to all the men who have discussed these papers. All otologists know the problem that meningitis presents and the heartbreaks with which we approach it. I have been much interested in the problem of meningitis for many years. I gave up the procedure which I advocated many years ago because of the utter failure which all my attempts at surgery met; the cases died, and more or less at the suggestion provoked by the observations of Dr. Eagleton, and inspired by the success of Dr. Dandy, in Baltimore, and Emerson, of Boston, whose cases I have studied, I came to the conclusion that they proved that the method of draining the meninges is feasible and the after-effects were good. As Dr. Eagleton said, in his fracture cases he feels that the line of fracture is the route of the infection. Dandy had wonderful success where the dura was infected secondary to operation; and the patients when tapped showed they had meningitis, and they got well under drainage. Our otologic cases died; so therefore it was necessary to move the operative procedure onward, and since then we have had recovered cases.

Now, it is very hard to translate a clinical picture entirely to paper. We do it as definitely as we can, but the same findings sometimes occur in dissimilar cases and you cannot see why two cases with the same findings are not the same; but there is a difference, in my opinion, and I present this as a working hypothesis, not as anything on which to take a final stand.

I have two cases—one to which Dr. Reuben referred, and which does not belong to this category; and one in which I did a cisterna puncture and the patient died; but the pathologist said definitely that a meningitis resulted—in that instance—from an ethmoiditis that had been overlooked; and cerebral drainage in that case was inadequate. Just as I have endeavored to show, the drainage of the cisterna magna was miles away from the route of the infection.

I cannot answer all the questions that Dr. Eagleton brought up, but will take them up in future work. In reference to Gradenigo's syndrome, the one that refers to an otitic disease is a localized meningitis; and if the dura is swollen and infiltrated by round cells, that is technically meningitis, and inflammation of the meninges is a meningitis technically. I hope that when the gentlemen read the paper at their leisure they will see that many of the points they have disussed are there taken up.

Replying to a query about magnesium sulphate enemas: we use that as a routine and by rectum because it lessens the intracranial tension. It is an observation made by all cranial surgeons, that if you prepare your patients that way they will have less tension. In cases where we have done cisterna drainage we have no herniation at all; and in cases where we drained the cisterna minor we have had no herniation.



Jonathan Wright

Jonathan Wright was born February 2, 1860, in Springboro, Ohio, and died May 26, 1928, in New York City.

Medicine, and Otolaryngology in particular, mourns his loss. One of the most scholarly scientists of our time has passed away and those who knew him will never forget the effect that he, through his influence, had on our present conception of scientific otolaryngology. He confined himself not only to his specialty, but also to problems of pathology, evolution, philosophy and the history of medicine.

He retired from active practice and work in 1915 but maintained his connection with the medical world through prolific scientific writings and medieval medicine and philosophy.

Jonathan Wright graduated from the College of Physicians and Surgeons, in New York, in 1883 and shortly afterward married Miss Susan Choate. He followed the practice of his profession in New York City until 1915 and maintained affiliation with many of the largest organizations in that city.

Doctor Wright was the author of two textbooks, "History of Laryngology and Rhinology" (1902), and "Diseases of the Nose and Throat" (1914). This latter work was done in conjunction with Dr. Harmon Smith. Dr. Wright also contributed the chapter on "Sinus Pathology" in Sluder's textbook, "Headaches of Nasal Origin". Some 125 scientific papers on divers subjects appeared by Dr. Wright, written from 1888 to 1928.

All of his work and all of his writings portray the charming personality for which he was so beloved. We are indeed unfortunate in the loss of Jonathan Wright.

M. F.